4

ON THE

PATHOLOGY AND TREATMENT

OF

ACUTE RHEUMATISM.

BEING THE LUMLEIAN LECTURES DELIVERED BEFORE THE ROYAL COLLEGE OF PHYSICIANS IN 1853.

BY

JAMES ALDERSON, M.D., F.R.S.

SENIOR PHYSICIAN TO ST. MARY'S HOSPITAL, AND FORMERLY FELLOW OF PEMBROKE COLLEGE, CAMBRIDGE.

LONDON:

JOHN W. PARKER AND SON, WEST STRAND.

1853.

LONDON: PRINTED BY W. CLOWES AND SONS, STAMFORD STREET.

PREFACE.

If there be a subject on which no apology is needed for travelling beyond the beaten track of investigation, it is Rheumatism; for all who have explored those paths have acknowledged that they have not been led thereby to any certain issue, and that not even a hopeful prospect of any satisfactory conclusion has opened out before them.

The line of argument which I pursue in the following pages does not deserve, however, to be considered altogether new. It is rather the return to a simple train of investigation, which appeared to me to have been too hastily abandoned.

While I submit this short Treatise to the notice of the Profession, I am so far from conceiving the sub-

ject to be completed, that I have entertained some doubt whether it might not be more judicious to postpone the publication until further time and study should have afforded opportunity to extend the views propounded, and to treat the subject more minutely, as well as more comprehensively: but having already delivered the lectures before the College of Physicians, I conclude that it is best to publish them at once, and to reserve the completion of the subject for a future occasion.

CONTENTS.

ON THE

PATHOLOGY AND TREATMENT

OF

ACUTE RHEUMATISM.

LECTURE I.

Upon Acute Rheumatism so much has been said and written, that I feel it is an act of courtesy due to you, who are so obliging as to listen to a dissertation on an apparently exhausted subject, at once to relieve you of the fear that I may linger on details with which you are perfectly acquainted: I shall not therefore weary you with a history of the disease or lengthened description of its symptoms and various complications. But while it is an easy task thus to declare the subjects with which I do not intend to occupy your time, the statement of what I do propose to lay before you may, I almost fear, display a boldness bordering on presumption. Its aim and object will be to explore the cause as well as to describe the true nature of the disease.

I shall hope thereby to solve the difficulties of a pathological condition which has been involved in much obscurity. I shall occupy my First Lecture with a review of all the data on which I hope to establish my views.

Let me here apologize for the elementary character of the physiological details which I shall have in the first instance to bring forward:—To lay them down, by way of groundwork, appears to me indispensable. On the present occasion I shall only be able to deduce from these data a general outline of my proposition.

In the Second Lecture I shall go minutely into the arguments for and against it, and endeavour to test the truth of it by all the collateral evidence which bears on the subject. I shall also review the various hypotheses which have been received or proposed.

My Third Lecture will be on the Action of Remedies, with an endeavour to elucidate the truth of my proposition, by comparing the actual results of different, nay, conflicting modes of treatment, with those to be looked for supposing it true.

If we open the pages of any of the ancient authors, as, for example, of Paulus Ægineta, we cannot fail to admire the large proportion of truth which the acute observation of the ancients enabled them to deduce from those very slender premises which lay before them, devoid as they also were of means to pursue more than a very superficial research into the nature

and causes of the phenomena of any disease. On reviewing the modern works which have been composed on the same subject, we shall observe with surprise and regret, that though we possess an accumulation of ascertained pathological facts, and powerful mechanical and chemical agency for scrutinizing beneath the surface of organized structure, in an age, too, when there is no lack of learning, industry, reasoning power, or eagerness to investigate, the actual nature and causes of many diseases, and above all others, that of which I am now about to treat, remain as obscure as they were twelve hundred years ago.

It may no doubt be thought, that to advance such disparaging comparisons is easier than to prove them. I will therefore briefly quote a passage to the very point from Paulus Ægineta, and then read to you its parallel from the last-published book* upon the subject in this country; a work in which I rejoice to appreciate the intelligent industry with which all the axioms of the present day are luminously stated,

In the 78th section of his Third Book, Paulus Ægineta observes, "It is not the weakness of the parts alone which produces the affection of podagra and arthritis — since, in that case, the paroxysms would be unintermitting, inasmuch as the

* Dr. Fuller on Rheumatism.

weakness also is constantly present in the weak joints—neither is it a humour *only*, for the affection does not attack the joints alone—but both together—an unnatural humour and the feebleness of parts produce disease." If we supply the requisite distinction between gout and rheumatism—it will appear from this passage and its context, that Paulus Ægineta considered rheumatism to be the result of a predisposing weakness combined with a certain morbid humour in the system.

How exactly is the same idea, and nothing further expressed, only in more modern language, by Dr. Fuller, when he says, "That the rheumatic poison has a special affinity for the fibrous and serofibrous textures throughout the body, and fixes more particularly on those which are in any way subject to irritation;" or when he further speaks of "a poison engendered in the system by faulty assimilation, as being the cause of rheumatic inflammation;" or suggests (when treating of the heart affection), "that the heart being enfeebled by the withdrawal of its natural and necessary stimulus, becomes unusually susceptible of morbid influence and succumbs under the irritation of the rheumatic poison." Thus, although the more philosophical language of the moderns contrasts favourably with the rude expressions of the ancients, their ideas upon the subject, are, in fact, the same. In both, any real explanation is evaded by taking refuge in

indefinite expressions which convey no lucid meaning to the understanding. There is no more precise revelation in the words, morbid influence, rheumatic poison, a poison engendered in the system, than would be conveyed by saying, that beyond such and such processes, some unknown agent must be referred to as the primary cause.

I have alluded to the slender premises possessed by the ancients, on which to frame a theory, as inducing wonder that we should have advanced so tardily towards that result after the immense amount of physiological discoveries which have since been made; but I conceive it is the very riches of the new revelations on the nature of texture and chemical analysis, which seduce the moderns, rather to labour like gold-finders in accumulation of their treasures, than to pause and use the whole weight of intellectual power in turning those treasures to account.

The Germans have been especially diligent in examining and describing these newly-acquired treasures of knowledge; and in so doing they have caught at many truths, which by a further combination of all the facts which bear upon the case might have been fully elicited.

My intention at present is to examine all the facts bearing on our subject which modern science has furnished to us within the last few years, such as the state of the blood, the secretions, the tissues,

&c.; to state the characteristic symptoms for which we are to endeavour to account; and from these materials, arranged and collated, I hope to form a chain of reasoning, of which the links shall hang so closely and aptly together as to support the weight of a general inference, applicable to the explanation of the subject before us.

Though I propose to avoid detaining you by unnecessary detail, yet we are obliged, merely to identify our subject, to give a short description of the external appearances of that form of the disease which I intend to treat of; for there are so many counterfeits, so many relatives which bear the name of rheumatism, that the individual we especially mean ought to be distinctly understood.

The few prominent symptoms which it will be necessary to specify in order to identify acute rheumatism are these:—It sets in with general lassitude, shiverings and heat, sleeplessness, quickened circulation, strong rapid pulse, scanty secretion of high-coloured urine, dry tongue, and thirst. The joints are then attacked by pain, heat, redness, and swelling; the tongue is covered with white slimy fur, tipped with red, and the surface of the body is bathed in perspiration of acid odour; the slightest motion of the joint exacerbates the pain, and the smaller joints display more redness and general swelling than the larger.

There is a period of increase and of decline of pain in the parts attacked, the average turning point being about three days from the first accession. One joint only may be attacked, or several simultaneously; and the symptoms frequently abandon one or all the joints attacked, and reappear in other joints, or in some vital organ within the chest.

Effusion takes place in the course of the disease, both in the joints and in the chest, which progresses sometimes so insidiously, that it is not perceived until an alteration of the form renders it apparent. Such, then, is a general outline of the external symptoms of acute rheumatism. We shall now transfer our notice to the state of the blood and the various secretions.

In acute rheumatism the blood is found to consist, in 1000 parts,* of

805·4	parts	water.
6·7	,,	fibrine.
101·0	,,	blood corpuscles.
86·0	,,	residue of serum.
999·1	,,	

The solid residue of the serum gave an average of inorganic constituents 7·9 in 100 parts.

* Andral and Gavarret.

As in healthy blood the component constituents are

790	parts	water,
3	„	fibrine,
127	„	blood corpuscles,
80	„	residue of serum,
1000	„	

it is apparent that in rheumatism fibrine is present in more than double the usual proportion.

The above tables are furnished by Andral and Gavarret as the mean result of forty-three analyses in fourteen cases of acute rheumatism.

By still more recent investigations the proportion of fibrine in healthy blood has been ascertained to be considerably below 3 parts in 1000. The excess, therefore, in rheumatism may be assumed to be even greater than it appears by these tables. The blood corpuscles are reduced 26 in 1000 parts below the healthy standard, and the water is increased above that standard. It will be borne in mind that I am now giving a bare statement of prominent facts, reserving for the present all more minute particulars. I proceed in the same way to notice the analyses of the secretions of the kidneys and the skin, as the composition of these fluids, at least of the blood and the urine, present a marked difference between the healthy and the diseased state, which lies within the scope of examination.

In the first instance, I wish to observe for a moment on the circumstances under which the analyses of the fluid can be effected.

In regard to the blood:—There is no difficulty in obtaining a correct analysis of healthy blood, because the necessary quantity can be obtained from a number of individuals, and the aggregate of analyses can be taken; but in disease, when the blood of one individual only can be available, a sufficient quantity cannot be taken to examine all the various constituents separately. This accounts for the incomplete analyses as regards the minor constituents.

In regard to the urine:—There is a great discrepancy, even by the most able chemists, in the analysis of healthy urine—a discrepancy which arises according as they use fluid which has been passed at particular times of the day, or that which presents the average of twenty-four hours, the nature, as is well known to you, differing according to the time.

In regard to both fluids, I would observe that no very exact inferences can be deduced, unless they be from analyses, made by the same individual, of both healthy and diseased fluids.

In regard to the urine in acute rheumatism, the principal results arrived at, from a number of quantitative analyses, consist in the preponderance of uric acid and of urates, chiefly urate of ammonia, over the proportions found in the healthy state. The urea is either increased or at the normal average.

The uric acid is always absolutely increased; in health the proportion is usually as small as ·61 in 1000 parts: in rheumatism it is stated by Simon* to be as high as 1·70 in 1000 parts; the salts are absolutely diminished,—especially the chloride of sodium; the sulphates are near the average of health, or but little below it. Free phosphoric acid and acetic acid have also been found.

The urine has a strong acid reaction; the colour is dark-red, as in inflammatory diseases generally: albumen was detected by Becquerel in seven cases out of eighteen which he investigated. During convalescence the urine is similar to that which is observed in anæmia.

As far as I can ascertain, the perspiration in rheumatism has never been quantitatively examined, owing to the difficulty of obtaining it in sufficient amount. It has a strong acid reaction. The odour is acid. I have myself attempted to make an analysis from a portion collected on lint, which appeared to be in sufficient abundance for an experiment. The lint, which had been thoroughly soaked with the perspiration, was introduced into distilled water with carbonate of lead; but when tested with hydro-sulphuric acid, the lead salt was not found in sufficient quantity to give a colour, which would be indicated by a seventieth part of a grain in a gal-

* Animal Chemistry.

lon,—a proportion quite inadequate for the purposes of analysis. The result proves the minute quantity of acid which was contained in the secretion. Part of the acid, no doubt, had disappeared, in consequence of its very volatile nature; but even with this allowance it is obvious that the original amount must have been very slight to have left scarcely a trace behind.

Simon, and Prout, and Berzelius, and some French chemists, have stated that the free acid of the perspiration, as it exists generally, which is present to an increased degree in rheumatism, is lactic acid. The more recent examinations of Liebig prove that no lactic acid is ever to be detected in the animal fluids, with only one exception, the juice of the flesh, which may hardly be called a fluid. Simon is of opinion that where there is an acid smell, acetic acid is always present. He would, consequently, refer that acidity which characterizes the odour of the perspiration in rheumatism to the presence of acetic acid.

Experiments to ascertain the solid constituents of the perspiration in health have been made with sufficient success to show that they consist of salts and extractive matters, of which the principal ingredient is the chloride of sodium, amounting to about 3 parts in 1000; the rest is simply water.

I wish earnestly to invite your attention to one evidence which is deduced from the examination of

these various fluids—the blood, the urine, and the perspiration—viz., to the remarkable similarity which obtains in their conditions, especially in that of the blood and urine in true admitted inflammation, and in acute articular rheumatism.

In regard to the blood, from a number of cases of well-marked inflammation, Messrs. Becquerel and Rodier have observed the following results, viz.:—

	Water.	Fibrine.	Blood Corpuscles.	Albumen.	Salts.
In inflammation - -	791·5	5·8	128·	66·	6·438
In health - - -	779·	2·2	141·1	69·4	6·8

The most striking modification to be observed in these results is the increase of fibrine in inflammation: and the same modification has been ascertained by all the most able chemists, who have investigated the subject. It will be of consequence, however, to bestow some attention on the other deviations displayed from the natural standard, viz., the diminution of the blood corpuscles and albumen.

I append a Table, which displays in one view various analyses of the blood in health, in inflammation, and in rheumatism, by several of the first continental chemists.

	Andral and Gavarret.		Becquerel and Rodier.		
	In Health.	In Acute Rheumatism.	In Health.	In Inflammation.	In Acute Rheumatism.
Water	790	805·4	779	791	798
Fibrine	3	6·7	2·2	5·8	5·8
Albumen	-	-	-	-	66·9
Blood Corpuscles	127	101·1	141·	128	118 7
Residue of Serum	80	86	69·4	66	-
Fat	-	-	1·742	3	-
Salts	-	-	6·8	6·438	8·1

	Simon.	Rindskop.	
	In Acute Rheumatism.	In Acute Rheumatism.	Second Bleeding.
Water	801·5	809·973	-
Fibrine	6·321	4·652	5·856
Albumen	100·54	166·954	-
Blood Corpuscles	74·56		
Residue of Serum	-	-	-
Fat	3·15	-	-
Extractive Matters and Salts	11·86	18·44	-

In regard to the urine, which, being secreted from the blood, would be expected to be placed by disease in a position relative to that of the blood itself, it is found in general inflammation to be darker than usual; it has an acid reaction, and usually a high specific gravity. The quantity of urea is not much changed, but the uric acid is very much increased. The salts are always diminished, especially the chloride of sodium.

It is therefore apparent, as far as these secretions are concerned, there is no essential alteration of component parts, in consequence of disease, which

can be especially attributed to, or identified with rheumatism.

In making these comparisons between the blood and secretions in general inflammation, and the blood and secretions in acute rheumatism, we are struck with the great similarity in the constitution of these fluids in both cases.

From this identity of change, I draw a conclusion that what are called two diseases, are so far identical in character, that inflammation may be considered as the genus, and rheumatism as the species; and it will be my task hereafter to trace the nature of those particular circumstances which induce the general disease to pass into the special. For the present we pursue our observations on the general inflammatory type.

The principal deviations from the standard of health seem to consist, in regard to the blood, in the excess of fibrine and the diminution of blood corpuscles. In regard to the urine, in the excess of uric acid and of urates: could we determine whence these changes arise, how they proceed, and how the several constituents are formed in the organism, it would be an important step towards elucidating the precise nature of general inflammatory disease, and of that particular type which is called rheumatism. To trace the actual formation, or course of production, of the normal constituents of the blood, would materially advance the inquiry into the nature of

the diseased condition. Perhaps the most important of these constituents is the fibrine. In its natural state it appears to be in a grade of development beyond the albumen or the blood corpuscles. In a sort of sense, it may be considered as more approaching to an organized state, prepared or ready elaborated to form part of the solids of the body.

Although, by analogy with the development of the egg of oviparous animals, we must conclude that fibrine is originally formed from albumen, since the egg contains nothing but albumen and fat, and from those alone, assisted by oxygen, admitted through the pores of the shell (a process analogous to respiration), all the organs of the future animal are produced: yet there are pathological considerations which indicate that it is not *directly* or *consecutively* by the expenditure of albumen that fibrine is generated. It is stated by Robin* and Verdeil that it is formed from albumen, by what they term isomeric catalysis: this suggestion is, however, no more than bestowing a new name on the process of conversion. The elementary composition of fibrine and of albumen is well known to be identical; therefore, to assert by a new phraseology merely that they are the same and in contact, exerting a presence action, adds no explanation of

* Traité de Chimie Anatomique et Physiologique, Paris, 1853.

the process. If it be true that the fibrine is formed directly from the albumen, we are unacquainted with the conditions which govern its change into that specific state. The slight diminution, however, in the relative proportion of albumen in inflammation, corresponding to the large relative excess of fibrine, leads us to discard this idea, and to conclude that the fibrine does not come, directly at least, from this source.

There is, however, a more tenable supposition founded on pathological observation. It has been remarked by Andral, that the formation of fibrine is quite independent of the condition of the individual attacked, since it occurs in typhoid fever, in chronic disease, and even in anœmia, on the supervention of inflammatory disease. Fibrine has also been observed to exist in excess in the blood during an attack of inflammation, which had been set up in consequence of an extensive burn; and it is known to practical men, that laryngitis, which consists in, or is accompanied by, fibrinous exudation on the lining membrane of the larynx, is the most usual termination of burns about the throat in children. It appears, therefore, that there are *various ways* in which fibrine in excess may be produced. If we can trace one with any degree of precision, and especially that one which is most apposite for my purpose, we shall have gained a point. Whatever the cause may be,

the first effect produced is, to increase its relative proportion in the blood; the second, to cause a retardation of its coagulating power; and the third, to enable it, or to induce it, to be exuded on the free surface of membranes, and in parts of the body not proper to it, *i. e.*, where it should not exist in a healthy state. In other words, let the conditions under which fibrine in excess is formed be what they may, they retard its coagulation; they induce its presence in parts of the organism which is foreign to it; they permit its exudation from the vessels which contain it into serous or mucous cavities, or upon the free surfaces of the membranes forming them, where it becomes organized, or at least passes into a state of more elevated organization.

It has been ascertained, as we have already shown by analysis, that the amount of fibrine in inflammation always varies inversely as the mass of the blood corpuscles.

It is on this pathological fact that Professor Simon, of Berlin, has founded his opinion of the origin of fibrine. He supposes fibrine to be formed by the active metamorphosis of the blood during respiration, *i. e.*, during the process of the consumption of oxygen by the blood corpuscles.

The respiratory process is known to increase not only the plasticity of the blood, but also the quantity of fibrine. On the other hand, the amount of fibrine is diminished in blood which is not efficiently

brought into contact with oxygen. If, therefore, blood be more rapidly and more frequently passed through the lungs, it becomes more highly fibrinated by appropriating to itself more oxygen. The converse of this proposition is no less established, viz., that blood, which stagnates in vessels, loses fibrine; for the fibrine is then consumed, and, in consequence of the stagnation, no fresh supply of oxygenated blood can be furnished. I invite especial attention to this last-stated part of the proposition, because I shall have to use it presently as the foundation on which to build an important part of my argument.

Simon further supposes that the *nuclei* of the blood corpuscles possess a chemical character which approximates them to fibrine, and that this important element of the blood (the fibrine) is formed from the *nuclei* by a metamorphic process, accompanied by the absorption of oxygen and the evolution of carbon. On the other hand, many high authorities deny the very existence of nuclei in the blood corpuscles.

In the healthy condition, the relative proportions of the blood constituents are maintained in an equally-balanced state; but when the circulation is accelerated, and consequently the metamorphosis of the blood corpuscles quickened, the mutual action between the blood and oxygen is increased; more blood corpuscles are consequently, during that acceleration, brought into contact with the air, and so, in any given time, more are consumed than would

be so in the healthy state. On the other hand, if the circulation is impeded in any part of the body, and the blood is prevented from receiving its due supply of oxygen, the metamorphosis is retarded and rendered incomplete; matured blood corpuscles which are approaching the state of solution are not dissolved, and there occurs an accumulation of colouring matter, especially of hæmaphæin.

Having considered the formation of fibrine generally, we must now regard its formation in excess under the influence of cold; and we must first briefly refer to the connexion between animal heat and chemical change, the point which we aim at being to show that a reduction of temperature interferes with what are called the vital processes—meaning those chemical changes, guided by a vital power, which take place at the periphery. It has long been established that the heat of the body is produced by the consumption of oxygen and the evolution of carbon in its lungs; but this cannot be the only source of heat. We are practically aware that the lungs cannot be the only seat where heat is generated, because in that case they would be hotter than any other part of the body, which they are not; and we must infer that the production of heat takes place on the whole peripheral surface of the body, where, although the action will not be effected in precisely the same mode with that in the lungs, yet as heat is always generated by chemical action, there remains

no doubt that some must be evolved by the processes of nutrition and secretion, and by the absorption of the disintegrated tissues. There are other modes in which heat is produced, to which we may just allude, although they bear less on our subject than the highly-important processes in the peripheral system of which we have just spoken; for instance, in the transmission of solid bodies through moist membranes,* and in the contraction of muscular fibre. In the last instance it is presumable that in the internal organism, the heart and the intestines are always, to a certain point, performing their part in keeping up the supply of heat. The generation of heat by the passage of solid bodies through moist membranes may, perhaps, be regarded as a part of that very change which takes place in the peripheral system.

Direct testimony to nearly all the sources enumerated is to be found in the increased temperature in inflammatory diseases, in which the blood, circulating more rapidly, passes quicker and more frequently through the lungs than in health. Whereas in morbus cæruleus, in the low temperature in chlorotic patients, in the torpid circulation of the aged, and in many other pathological conditions, negative evidence may be perceived of the several sources by which heat is generated, interesting to trace, but too

* Poullet, Annales de Chimie et de Physique, vol. xx. p. 141.

parenthetical for our purpose. The hybernation* of animals bears too much on our argument to be so slightly hinted at, for it is a strong corroboration of the fact of the mutual dependence of animal heat and chemical change upon the blood. In this peculiar state, respiration and circulation become slower, and it is found that the blood does not contain its usual amount of fibrine and albumen. Thus a proof is afforded that when oxygen is not brought into connexion with the blood, fibrine is formed in smaller proportion.

We have thus seen, that the presence of atmospheric oxygen is absolutely necessary for those changes in the corpuscles of the blood which take place in the lungs. We have also seen that one result of those changes is the development of heat. Another, the conversion of a portion of the corpuscles into fibrine. These changes may be produced, as supposed by Mülder, by the oxidation of the protein compounds : there is a controversy on this topic ; but whatever be the actual process, whether Mülder's theory of it, or any other be the correct one, the result is, distinctly and undoubtedly, the production of fibrine.

I have now to show, that under the influence of cold, or of cold-taking rather, the process of conversion into fibrine is accelerated, and that that

---

* Simon's Chemistry (Sydenham Soc.) by Day, vol. i. p. 145.

constituent of the blood is made to be present in an undue proportion. The generally-admitted cause of rheumatism is, the influence of cold upon the surface of the body, together with a sudden admission of cold atmospheric air to the blood circulating through the air-cells of the lungs. This injurious influence of cold on the surface is supposed to be greater when the cold is combined with moisture, acting by its conducting power and not by evaporation. It exerts more energy if applied to the organism at a time when the temperature of the body is elevated beyond the natural standard, or when the surface is moistened by perspiration.

I will consider first the effects of cold on the blood in the lungs. In most cases, persons who are the recipients of this injurious consequence of cold have been previously exposed to an atmosphere vitiated by an excess of carbon; when, having suffered a temporary privation of oxygen, the organism accepts, with renewed avidity, that necessary agent for the vital change in the blood. The blood corpuscles appropriating the oxygen in excess, the binoxide or the tritoxide of protein of Mülder, or what we call fibrine, is formed in larger proportions than is compatible with the healthy state.

The word cold, as applied to cold-taking, must not be understood in the more usual sense of depression of temperature, although that actual de-

pression is included in the term. It is meant to imply, cold produced by some sudden changes in the atmosphere, as, I believe, through electrical causes.

Although casual opportunities occur for the influence of sudden vicissitudes of temperature, especially when, as just observed, following after exposure to a carbonized atmosphere, yet the vast number of inflammatory attacks take place in certain peculiar atmospheric conditions when the atmosphere may be considered in a chemically-excited state, or, in other words, when the ozone of Schönbein is present.

It is obvious that oxygen, in this state, would act with increased power for the over-production of fibrine. Whether we may attribute all inflammatory change on the blood by the reception of the excess of oxygen to this chemically-excited state of the atmosphere is at present hypothetical; but the evidence, which is increasing daily before us, disposes me strongly to believe, that in this consists the exciting cause of the over-fibrinated state of the blood which constitutes inflammation.

Such reference to atmospheric condition, together with this mode of its influence on the system, suggests a solution of the difficult question of the nature of epidemic disease.

We may view the fibrine in this inflammatory state as having received a forced development into a more advanced stage towards organization; or, in other words, an approach to a more exalted state of

vitality; too exalted perhaps for the offices which it has to perform.

We have now to refer to the process which takes place during the abstraction of heat through the conducting power of a cold and moist atmosphere, suddenly applied to the surface of the body.

It is apparent that fibrine and albumen, not being found in the various secretions of the body during health, must, in the normal state, be expended in the processes of nutrition, which processes take place in the capillary system, by virtue of a power there resident, to assimilate these constituents of the blood: when these processes are interfered with by the abstraction of heat at the surface, and by the constricting effects of cold on the extreme vessels, fibrinated blood then passes through vessels, the cells of whose capillary network ought to be employed in selecting and assimilating the nutritive parts of the blood; those vessels then permit the blood to pass through them and thus fibrine in excess, unconsumed in the peripheral system, is transmitted to the veins. So far upon the action of cold on the surface. We have already seen, that chemically-excited oxygen applied to the extreme vessels of the lungs produces fibrine (a mere chemical act): we have also seen, that by the stoppage of the processes of nutrition, this fibrinated blood is passed by the extreme vessels into the veins and so to the heart: that the presence of this fibrinated blood stimulates the heart to undue

action,—that the consequence of this accelerated motion of the heart is a more rapid transmission of blood through the lungs,—and that that more rapid transmission causes again a still increased rate of consumption of corpuscles in the formation of fibrine. It plainly follows, therefore, that if in this condition no supply takes place, by nutriment, to replace the consumed corpuscles, the corpuscles will be diminished in their absolute quantity, as well as in their relative proportion to the other constituents of the blood. This takes place in inflammation, in which we have shown that there is a deficiency in the blood corpuscles.

If we can assign no explanation of the reason or way in which the heart is so stimulated by abnormally-fibrinated blood, we can, at all events, attest the fact by positive observation as well as by analogy. We know, that in all inflammatory diseases, excess of fibrine occurs, and that there is a quickened circulation. On the other hand, in typhoid fever, we know that there is a deficiency in fibrine, and that the circulation is torpid.

To recapitulate what has been stated, I give a summary of the various points insisted upon. We have fibrine formed in the lungs by the sudden application of chemically-excited oxygen: we have simultaneously a stoppage of the processes of nutrition at the periphery: we have a super-oxygenated blood transmitted to the capillaries, and by those capil-

laries (in consequence of the disturbed relation between them and the blood, depending on the abnormal proportion of the constituents), that super-oxygenated blood transmitted to the right side of the heart: we have this super-fibrinated blood stimulating the heart to increased action, and we find this action further increasing the fibrine by means of a quickened circulation, while at the same time the continued want of the consumption of the fibrine at the periphery still promotes its accumulation.

This statement, I must observe, includes nothing hypothetical. The facts are all fully borne out and received, and nothing has been added but to connect them in one comprehensive view of cause and effect. The state we have described obtains in general inflammation, and to it all the usual symptoms may be referred. Thus we find that in inflammation all those organs which depend for the due performance of their functions upon the normal constitution of the blood are disturbed. There is increased heat, the circulation is quickened, and the secretion from the kidneys is scanty and faulty, &c.

Having traced, to the best of our ability, the pathology of the blood and secretions in common inflammation, we have next to inquire what are the external symptoms which characterize acute rheumatism, in order to distinguish that disease from the general inflammatory state. What, then, is that

which gives to rheumatism its specific character? It is not to be found in the state of the pulse, or of the tongue, or in the constitution of the blood, or in the nature of the urine, or even in the acid state of the perspiration. The pulse is identically the same; the tongue in both cases is coated with white fur tipped with red; although authors refer to a particular state of the tongue, I have never distinguished any real peculiarity. The fluids display a remarkable correspondence. In the blood, the chief characteristic is, equally in both diseases, that excess of fibrine which we have been examining. The urine in both has the same high colour, scanty quantity, and identical constitution, having excess of urates and deficiency of chlorides. The acid state of the perspiration, which has often been insisted on as a characteristic of rheumatism, differs from that of inflammation, not in the nature, but in the degree of the acidity, and perhaps in the greater diffusibility of the odour.

The distinctive character, therefore, lies in none of these; but rheumatism differs from common inflammation in the superadded symptoms of pain, heat, redness, and swelling of the joints, and in that which has been formerly called "metastasis," which consists in a sudden and complete change of the seat of the local affection. In this so-called "metastasis," pain and redness suddenly desert a joint, move from one joint to another, or seize a vital

part. This, which has been termed also "shifting," or sudden recession and translation from one locality to another, is of all these characteristics the most tangible and prominent. Therefore the pain, heat, redness, and swelling of the joints, and the so-called "metastasis," are the chief subjects to be considered: and for this purpose we must first review the structure of the joints, and then refer to the nature of the tissues, together with the mode by which blood is supplied to them.

We find the structure of the joints to partake of the nature of a hinge, and to be covered first by a synovial membrane, which either secretes a lubricating fluid, or permits the fluid to escape by exosmosis from its free surface. Beyond, or without the synovial, we find a binding membrane, which gives strength to the joint: these membranes consist of white and yellow fibrous tissues, intermingled and interlaced with each other in various proportions, of which the inelastic white fibrous tissue constitutes by far the larger. The tendons and aponeurotic sheaths are formed of the same fibrous tissues.

The ultimate distribution of vessels to these tissues is not very distinctly ascertained. It is, however, stated by the latest authorities* on microscopic observation, that the capillary vessels which are

* Queckett on Histology, p. 132.

distributed to these tissues are not so numerous as in the areolar tissue, and the vessels, connected by branches more or less oblique, run a straight course between the bundles of the white fibres, not amongst the white fibres themselves. The number of the vessels in a tendon is very small compared with that in a muscle.

By this arrangement, the capillary vessels in the fibrous tissues are, in the natural state of necessity, compressed within and between the bundles of inelastic white fibrous tissue, and are, in a manner, bound down by it.

This is a distribution of vessels and tissues especially liable to become the locality where circulating fluids may be obstructed; and the inelasticity of the white fibrous tissue is eminently calculated to increase the dense packing of the accumulating fluid. The combination of parts is precisely such as would offer obstruction to the transmission of blood which is not normally constituted, and the resistance which is offered to distention is calculated, not only to create obstruction, but to cause pain.

The vessels of the synovial membrane, which forms the capsule of the joint, observes Queckett,* are found thrown into a series of processes like villi, which are largely supplied with vessels remarkable for their tortuosity. These villi project into all the

* Histology, pp. 156-7.

parts of the cavity of the joint, and the vessels are supposed to pour out the synovia: they may therefore be viewed as synovial glands; and similar processes which no doubt secrete a fluid somewhat like synovia are found within the sheaths of tendons.

When the vessels of the synovial membrane are examined by a higher power (250 diameters), the capillaries will generally be found to terminate in loops;* but the synovial membrane extends some little distance beyond the vessels, and the non-vascular portion is covered by epithelium. This rich superficial network is not found amongst the tendinous fibres of tendons but only in the sheath and in the synovial membranes.

This displays the great facility with which fluid may be poured into the capsules of joints and sheaths of tendons.

Henlé remarks on the subject of rheumatism, with great reason and discernment, that we ought to reason backwards in attempting to explain the symptoms which we witness. It is plain that in order to do so, it is indispensable to display clearly all the facts on which we mean to fall back to complete the train of argument. For this purpose, I have stated as briefly as the subject will admit, whatever in the present state of science may be safely depended upon, in respect to the constituents

* Toynbee, Phil. Trans., 1841.

of the blood; the changes produced in it by the action of the atmosphere; the chemical composition of the secretions; and the peculiar structure and mode of supply of blood to those localities which are the first attacked by the disease. In doing so, I have been obliged, even at the risk of being tedious, to detail a variety of facts which are perfectly well known, and should any of them be less familiarly understood, there are acknowledged authorities to support their accuracy. These being fully sufficient for the purpose in view, I shall produce no statements either new or unsupported to assist us in our present inquiry into the true character of the causes of all the symptoms which we witness in rheumatism. We have only now to arrange these facts in their proper order, and throw their collected light upon a subject which has so long remained obscure, —and if we do not wholly reveal the truth, I hope we may at least clear the road for future investigators.

We have seen that in rheumatism, exactly as in common inflammation, the blood is in a state unusually charged with fibrine; the blood so changed being more plastic than in the healthy condition; and also, that the proper relation between the vessels and the blood is disturbed by the altered conditions of the constituents of the latter. We know, from microscopic examination, that wherever there is local inflammation, there is a retardation of the

blood in the minute vessels and in the capillary network. Now, as we have observed, that the vascular supply of the fibrous membrane of the joints is most delicately and minutely distributed, the nature of the organ, which partakes of the character of a firm resisting bandage, causing that vascular supply to be distributed in more minute vessels than in the other tissues of the body, it is a plain conclusion that the plastic fibrinated blood is likely to be delayed in its transmission through the minute calibre of those vessels more easily than in other parts: and when we take into account at the same time the velocity of transmission of blood from the heart by its stimulated action, we have a reason, coupled with the delay in the vessels at the joint, to account for the very considerable state of distention in them. By the continued existence of the obstructing causes within the joint and by the pressure without, this delay gradually becomes a stasis, more or less complete; and if here we take into consideration, that it has been shown by the microscope that such a stasis does, in common inflammation, actually occur, in parts, where the vessels are not bound down by fibrous tissue, there can be no difficulty in supposing that such a stasis would take place, in a far greater degree, where those binding tissues exist. If this combination of parts not only is calculated to offer resistance to transmission, but is so to an extent not to be found elsewhere in

the organism, a reason appears why these tissues and this locality of the joints should be selected as points of attack; or, rather, why the blood being in an abnormal state, should there become first obstructed. The same tissues, it is true, do exist elsewhere, and are to be found in the heart, &c., but nowhere do we meet with that same resistance which belongs especially to the formation of the joints. This remark would lead to the subject of extension to the heart; but we must defer that for the present.

We deduce from this a reason to account for the seizure of the joints in preference to any other parts of the body, and we can account for the pain which is the consequence of the distension of vessels: we have still to explain the causes of effusion—the redness of the surface—the sudden change of the local seat of attack—and the equally sudden and final departure of some or all of the symptoms.

It follows in consequence of the distension of the extreme vessels that the thinner parts of the blood are forced through the coats of the vessels, either into the surrounding cellular tissue, or into the synovial cavity. When the fluid is of a purely serous character, as it almost always is in the joints, I believe the process to be simply mechanical, the fluid being poured out by exosmosis through the free surface of the synovial membrane. When small portions of fibrine are contained in the fluid, we must believe the cells of the secreting vessels to have been more or less

engaged; but I shall have to refer to this point in future remarks.

The redness which we perceive in rheumatism may be accounted for by considering that, when blood is delayed in vessels, and not submitted to the action of oxygen, the metamorphosis of the blood corpuscles does not proceed; and hence the corpuscles, arrived at a state of maturity, are not consumed, and consequently there is an accumulation of colouring matter, of hæmaphæin.

The so-called "*metastasis*," that most interesting and (because especially characteristic of the disease) most important symptom, which has hitherto evaded all satisfactory explanation, remains now to be considered. When the delay of blood takes place in the vessels, the fibrine is, as we have already shown, dissolved, and, in consequence of the ensuing stasis, no more fibrine can be supplied to the part from the general circulation; the fibrine is in the circulation in excess, but the existing stoppage hinders its approach. The solution of the fibrine, therefore, proceeds in that quantity of blood which is delayed and accumulated in the locality of the joint, and tends to restore the balance towards the normal proportion in that quantity of delayed blood. Blood, therefore, which has been accumulated by means of this stasis has been subjected to a sort of purifying process; and the fibrine, having become no longer in excess, the disturbed relation between the vessels

and the constituents of the blood in that particular locality no longer exists; the capillaries no longer resist the transmission, and a free passage for the blood is once more obtained. Hence the depurating process being complete, we may see a mode of accounting for the desertion of one point of attack.

The local attack—or, in more explanatory words, the process of obstruction, of accumulation, and of restoration of the balance of the constituents of the blood by a solution of the fibrine—has, by this time, it is true, caused a return to the normal condition of so much of the over-fibrinated blood as has happened to be clogged in the delicate vessels of the part last affected; but the general over-fibrinated state of the blood continuing to pervade the system throughout, and having experienced but very partial relief from the small quantity lately restored and depurated, it is naturally to be expected that other joints, either in turn or several simultaneously, may offer a similar obstruction to the abnormal blood, which will then and there go through the same renovating process.

All this supposition agrees with the known symptoms of the disease; and thus, that which has been considered as an occult and mysterious peculiarity, showing as if what has been called the "morbid influence" delighted in arbitrary and sudden selections and abandonments of its points of attack, may be regarded as a regularly consecutive and simple event, governed by one single law, and that law marked by

the same beautiful simplicity which may be traced through all the government of the universe.

In sketching this very meagre outline of a general idea of the nature of rheumatism, I have suppressed anything like argument, illustration, or consideration of the difficulties which at first sight may appear to attach to the proposition. These have yet to be placed before you; and in offering a suggestion, which wholly dispenses with all those favourite topics which writers on acute rheumatism are wont to dilate upon, I feel sure that my hearers are superior to the temptation of hastily rejecting it simply because it is at variance with preconceived ideas. At any rate they are too liberal and generous to do so, until they have heard the question more completely stated, and until they have listened with such patience as they can afford to the pleadings in its favour. In some points of view, scientific minds will, I feel sure, be disposed to regard it with hopeful indulgence: to them its simplicity will be its first recommendation.

Of all the objects to be desired in the study of pathology, a more comprehensive classification of disease is the very first. In all other sciences we have daily to acknowledge and rejoice that the simplicity of the laws of Nature becomes more apparent by every new discovery; and the innumerable phenomena which have hitherto appeared as so many isolated facts, are gradually being grouped and

classed, and referred to few, but very comprehensive, laws. That the departures from a healthy state of the human organism should also be referred to some more general laws, and recognised as modifications of one single disturbance, would be a marvellous stride in the progress of medical science.

Though I can only hope to offer an humble contribution towards such a result, I should rejoice in the reception of this view of rheumatic disease, even more as an approximation towards that great end than as a light thrown on a single pathological subject.

LECTURE II.

In our last lecture, after briefly detailing the distinguishing symptoms of acute rheumatism, I prepared the way for an examination into the nature and cause of the disease by investigating the state of the various fluids of the body in rheumatism, in health, and in general inflammation. It appeared that, in rheumatism, the deviation of the condition of the fluids from the state of health consists in the blood, in an over-fibrinated state, with defect in the blood corpuscles—in the urine—in the presence of uric acid, and urates and deficiency of salts—and in the perspiration in the presence of a volatile acid. I then showed the remarkable similarity which obtains between the abnormal state of these fluids in inflammation and in acute rheumatism, leading to the conclusion that these two types of disease stand to each other in the relation of genus and species.

In regard to the blood, as the chief deviation from the state of health appeared in both cases to be the excess of fibrine, we endeavoured to trace the origin of fibrine in the normal state, with a view to comprehend the formation of it in excess in disease, and also the modifications which it then undergoes. We reviewed Simon's theory of the formation of fibrine from the blood corpuscles, and showed the connexion which exists between chemical change and animal heat, and the manner in which loss of temperature, interfering with chemical change, results in an interference with the processes of nutrition. I then showed the influence of atmospheric oxygen in the blood generally, and that when chemically excited it causes the production of fibrine in excess, followed by the stimulation of the heart, and further production of fibrine by the increased rapidity of the circulation and the defective function of nutrition.

Having thus taken a comprehensive view of the state of the system in general inflammation, regarding especially the over-fibrinated blood as the most prominent characteristic, I next inquired what might be the essential difference between that state and acute rheumatism. Having already recognised an identity in the state of the fluids, we perceived that all the external symptoms of general inflammation were included in those of acute rheumatism, the latter disease having superadded to them local af-

fection of the joints, the remarkable incident of the so-called "metastasis," and the occasional subsequent extension of the attack to vital organs.

I then described the structure of the joints, the nature of the tissues involved, and their mode of vascular supply. I dwelt especially on the nature of the fibrous tissues, showing that, in conjunction with the structure of the joint, which includes power and facility of motion, the vessels in those localities are more delicate and difficult to be permeated than in other organs and tissues of the body. I then inferred, that the over-fibrinated, too plastic blood would find a ready obstruction in those parts, and that the increased velocity of the circulation arising from the over-stimulated heart would induce pressure in the stagnant blood, and a consequently increased accumulation. In this stasis, and the resistance of the vessels caused by the inelastic fibrous tissue, we found sufficient reason to account for pain, heat, redness, and swelling, &c., and the effusion appeared to be the natural result of this distension.

We then applied a fact which I had previously stated, viz., that blood which is not sufficiently brought into contact with oxygen loses fibrine, to the process going forward during the stasis, and argued, that the stagnated blood being there out of reach of atmospheric oxygen, and of further addition of fibrine through the medium of the generally circu-

lating mass of blood, was there going through a sort of depurating process, partially assisted by the effusion in respect of its mass, and also to a certain degree in respect of its richer constituents. The relief of the local attack I then referred to the restoration of the balance of the constituents of that portion of the blood which had been delayed in the part affected, and its consequently renewed power to permeate the vessels. I then observed, that though the quantity of blood obstructed had, by this means, been returned to the circulation in a renewed normal condition, yet the proportion which that quantity could bear to the general mass would be so small that the original defective state of the blood would remain but slightly mitigated; and the too plastic blood would still be in a position to suffer delay at other joints where the same anatomical structure presents the same liabilities, either in the joints or at the heart.

I propose that, in the present lecture, I should go more minutely into the subject, offer further arguments and illustrations, and endeavour to grapple with such difficulties as may present themselves.

Having been led by philosophical and chemical reasoning to feel something like confidence in the justice of this argument, I was glad to find a certain degree of support from Henlé, who, if he does not carry out the view, at least approaches the subject thus:—"The idea of an obstruction in the fine ca-

pillary vessels of the joints occurring in consequence of a faulty condition of the blood which stops the canals mechanically, must have presented itself as the cause of rheumatic joint-inflammation to every one who has reflected deeply on the nature of the disease." In the sequel, however, having proceeded to institute a comparison between this disease and phlebitis and pyæmia, hoping, by that means, to account for the "*metastasis*," and having found (as we might no doubt expect he would) that line of argument wholly inadequate to serve as an explanation, he then refers equally indistinctly to the complications, and dismisses the hypothesis without arriving at any conclusion in its favour. Thus he apparently recognises the idea of a stasis, although he fails to meet the phenomena which attend metastasis. That so acute an observer should acknowledge the correctness of all our premises till he arrived at that point of difficulty is abundantly satisfactory, because the solving of that problem is the very keystone of our present proposition.

Further, in regard to the degree of support that we may be able to derive from Henlé, I must observe, that while he shadows out that part of the statement which rests on the vascular supply, he quits the subject, by adverting to the want of sufficient knowledge on the arrangement of vessels in the ligaments of the joints, and invites attention to the subject. The evidence on this very important link

of the argument, which was not fully laid before him, is now supplied to us, for we noticed in our first Lecture the mode of supply of blood to these tissues, as given by Queckett, in his recent work on Histology.

Though not quite in place here, it will be as well to meet without delay a very obvious question, which may occur to many, and appear to them at the very outset to encumber our subject with a grave difficulty. The question may be thus proposed:—If the general state of the system, especially in regard to the constituents of the blood in general inflammation and in rheumatism be so nearly identical, that the difference consists only in the superaddition of localization in the joints, to distinguish the latter as articular rheumatism, why should general inflammation sometimes assume the type of rheumatism, and sometimes not? Or again, Why does not general inflammation always attack the joints?

Now, although I intend to meet this question fairly, and, I hope, successfully, I must observe that the point which I set out to prove was simply this: that in the over-fibrinated condition of the blood of common inflammation, calculated, as we have shown it, to find obstruction more easily in tissues, so especially formed as the binding tissues of the joints, a clear and sufficient cause is displayed for the nature of the seizure called acute rheumatism, without

calling in the aid of any specific or extraneous agency; and, moreover, that the same machinery which is sufficient to account for the attack, is sufficient also to explain the attendant phenomena of "metastasis," &c. In pointing out those particular tissues of the joints to be more likely to become the seat of the local affection than other organs of the body, I by no means imply of necessity that other organs, whose tissues are by natural formation less liable to obstruction than the binding webs of the joints, should not however be, from a vast number of circumstances, in certain individuals, at certain times, yet more impaired or more vulnerable than the joints.

My position is, that the general priority of vulnerability would lie in the joints, supposing all the organs to be in a sound and equally developed state, and that no adventitious localizing agency were to give a preference to another. A state of unbalanced relation of the blood and vessels in any organ does not render inevitable the occurrence of obstruction; it is a state of imminent danger, but as in other dangers the event may be suspended till some minute guiding incident fills up the measure of required impulse, and causes the catastrophe. The single drop may not be added to the fluid which seems to overtop the vase, and the crisis may be averted, but the imminent liability to the overflow has been the same, and the average results of many

such positions of the vase and fluid will go to prove the danger.

The guiding incidents requisite to give the last impulse to the joint attack may merely be some especially delicate vessel, some impairment by over-use, or many other casual circumstances, and the obstruction once begun, the original causes work forward towards a rapid accumulation. We know that in a thousand ways objects equally within the reach of one prevailing cause are subjected to special minor influences, by means of which the general cause becomes sometimes effectual, and sometimes not, while the last guiding impulse is often too minute to be perceived: were it not for this consideration, the same question which is applied to the joints, as compared with other organs, might be extended from particular joints to the rest. Why are not all the joints attacked, if the same constitutional derangement pervades them all? This question, you will perceive, is equally applicable to every possible theory that can be proposed. The answer has been anticipated. We cannot see the ultimate propelling power which causes the final catastrophe, at any one point; we only know that the dangerous condition of all the joints was such that the minutest possible impulse might be sufficient.

But to return to the immediate question. Why does not acute inflammation *always* assume the type of acute rheumatism? We repeat, that the lia-

bility, or even the eminently superior liability of one part does not include the necessity of its being always actually involved, but where the excessive liability exists, there a slight impulse may determine it; we only argue that when it does take place, the whole process of the disease can be accounted for. To insist that it must, without exception, take place in the joints, would be to deny that any modifying circumstances, whether from organic defect, accident, or habit, could exist in any other organs of the body to cause a preference elsewhere.

I do believe, as regards not only the joints, but also every other organ, that there are additional localizing causes which determine the point of attack. Some are already perceptible; some remain to be traced out; but, because these minute impulses remain yet partially concealed, there is no reason why we should therefore reject a general law, by which, after the localization is once determined, all the phenomena may be explained. But, leaving sight of those minute disposing circumstances which favour localization, I restate thus, what I have desired to insist upon, that the joints are disposed to be the seat of obstruction, or, in other words, that they have a *general* liability, from their natural formation, and, moreover, a *special* liability peculiar to themselves, in so far as that, when delay of the too plastic blood has once begun in any of the binding tissues, however small may be the extent of

that delay, it must go forward to obstruction in consequence of the peculiar resistance there existing, and so proceed to the complete development of all the rheumatic process: whereas, in other organs, where such peculiar tissues do not exist, a slight commencement of delay may be overcome.

In respect to the last propelling cause of localization, both in the vital organs and in the joints, I conceive that the subject will not be thoroughly solved to the satisfaction of every one until the entire range of inflammatory diseases are classed with more comprehensive reference to general causes.

There are individuals in whom particular joints are especially selected for attack. It is usual to attribute this selection to excessive states of exalted nutrition, but pursuing the idea that the particular nature of the vascular organization is the general cause of the obstruction to the abnormal blood, I should rather refer the choice of situation to some degree of damage done to the finer structure of the parts, the injured condition occasioning still more facility for obstruction than the mere anatomical peculiarity of the organ.

We should, for example, anticipate that which is really the case, that the foot, and ankle, and the knee, would be first in the list of joints attacked, since the nature of their duties, to carry the whole weight of the body, renders them especially obnoxious to wear and accident. The wrists and joints of the fingers

might also be expected to stand, as they really do, next in the number of localities, obnoxious to attack, for though less impaired by supporting weights and long-sustained exertions than the joints of locomotion, they are, notwithstanding, often subjected to forced and excessive action. The hip having less weight to carry than the foot and ankle, and being exempt from the uses of manipulation and labour, to which the wrists and fingers are applied, might be expected to be less frequently attacked, which it actually is. In reference to the hip, although considerably used in locomotion, there is a further consideration to account for the infrequency of the attack. The great power which, in the case of the knee, is afforded by the fibrous tissues, is in that of the hip-joint obtained by the arrangement of a profusion of powerful muscles, having a minute vascular supply, entirely different from that of the fibrous tissues. The elbow has neither weight to support nor any especial exertion of power imposed upon it. We might, therefore, expect to find it, as it is, lower still in the scale of joints attacked.

The large proportion of labouring persons attacked, whose occupations require excessive exertion of the joints, is an evidence that injury is a fertile cause of the localization of the inflammation. A review of the number of artizans who suffer, and of the localities which are attacked in each, connected with their methods of labour, would, I believe, from

the general observations I have myself been able to make, form an instructive exemplification of this cause of liability.

Immediate exposure to cold has been suggested as a means of inducing liability, but there is not much evidence to be derived from it in regard to the selection of one joint more than another; the elbow, which is a part as frequently exposed, if not more so, than other joints, would not, under this supposition, be found at the bottom of the list of the localities attacked.

It must not be overlooked that children are especially obnoxious to the disease, and that we cannot, by any supposition, refer the cause to over-exertion of their delicate joints, except in so far as exercise may be over-proportioned to their strength; but one fact bears upon the subject, viz., that they are actually oftener attacked with the general rheumatic type, but when they are so, they do not suffer in the joints so severely as adults, and the extension to the heart is more frequent.

The fact itself, that children are more subject to rheumatism than adults, presents rather a corroboration of the general view we have taken of the causes of rheumatism than a difficulty. The blood in children is in an over-fibrinated condition compared with that of adults; and therefore this fibrine-forming state of the system, which is habitual to them (for whatever purposes, it may be probably for

nutrition), may so favour the formation in excess as to cause in them a tendency to accumulate it more rapidly than adults when placed in the same circumstances.

It is no groundless surmise that children's tissues may, from their growing state, be less duly proportioned than those of adults to the performance of duties, which are exactly the same in the progressive and in the fully-developed state of the body. We know that parts of the body advance irregularly to maturity: witness the large hands and disproportioned limbs of young people. Hence we may infer a cause superior even to the damage of after-life, by which the general inflammatory type may more easily become localized in their joints, and by that localization assume the rheumatic form. That the heart should be the point of attack so frequently included may be referred to the known irritability of the heart in early life, which, by increasing its action, determines the affection to that organ.

The essential distinctive feature which characterizes rheumatism being, as we have already said, the local affection of the joints, we will pursue the inquiry, how far the views suggested, as to the nature and cause of the local attack, are borne out in applying the same principle of explanation to all the symptoms and incidents of the disease.

Relief from the intense pain, redness, and swelling, according to that explanation, may be considered as

a necessary sequence of the solution of the excess of fibrine, that solution having been fully effected by means of delay in the vessels, and in consequence of the absence of a sufficient supply of oxygen to the blood so delayed. The average period of the arrival of relief from the attack of each locality has been, by general observation, fixed at about three days, and this takes place without any artificial assistance. This general self-adjustment of the balance falls in with our statement in regard to the natural curative process. Under the head of treatment we shall have to consider how far that natural process can be assisted or accelerated.

The period of the duration of the acute febrile symptoms in rheumatism has a generally uniform limit, resembling that in the exanthemata, and the period may be regulated as it is in them by circumstances (though of a different kind), some perceptible and some concealed: the intensity of the symptoms has an ascending and descending scale or successive states of exacerbation, a maximum and then a decline: the time of arriving at the turning point is about fourteen days. Successive bleedings made during the presence of the disease give evidence that the relative quantity of fibrine bears a corresponding proportion to the febrile symptoms, both in increase and decrease, as well as in maximum point of excess. The period of duration of the febrile symptoms, and of the corresponding presence

of fibrine in excess, may, according to our supposition, be modified by the degree of self-relief which is afforded by the local affection.

I append a table of the analyses of blood in acute rheumatism, by Andral and Gavarret, after various bleedings :—

Venesection.	Day of Disease.	Water.	Fibrine.	Blood Corpuscles.	Residue of Serum.
FIRST CASE.					
1	4	797·1	8·9	109·3	84·7
2	5	796·9	9·8	107·5	81·8
3	6	812·5	8·5	95·4	83·6
4	10	820·6	6·4	93·5	79·5
5	25	769·7	2·8	117·9	89·6
SECOND CASE.					
1	8	778·8	6·1	123·1	92·0
2	9	780·9	7·2	120·7	91·2
3	10	788·0	7·8	112·8	91·4
4	13	799·0	10·2	101·0	89·8
5	17	813·9	9·0	89·2	87·9
6	28	826·2	7·0	83·8	83·0

Another important circumstance has also been noticed—that the more severe the symptoms are at the commencement of the attack, the sooner is that maximum point of excess of fibrine arrived at. The fibrine is, in fact, found to be produced more rapidly in proportion to the severity of the first onset of the disease; probably, it may be said, in proportion to the first exciting atmospheric cause. The increase up to a certain period is to be explained by the accumulation which must take place in con-

sequence of the fibrine not being used up at the periphery, and by the simultaneous process of its continued formation in the lungs. It would appear, however, that there is a limit to the formation of fibrine in excess, which may be thus understood. If nutriment is not supplied from which blood corpuscles must be elaborated to furnish fibrine—no more fibrine can be produced. The descending series in the scale of fibrinated blood will be in proportion to the severity of the self-relieving local symptoms.

In cases of relapse, the over-fibrinated state of blood does not apparently rise gradually, but jumps at once to a high point of excess, bearing still the same proportion to the febrile symptoms. These relapses occur from accidental exposure; which exciting cause has a more powerful influence over the constitution at a time when it has lately been in an abnormal state, for blood which has recently been in an unbalanced condition is more easily excited to a similar state by slighter causes.

An illustration of the subject may be borrowed from what takes place in chemical action. We know well that changes which are readily produced in extemporaneous solutions, cannot be effected with the same solutions if time be given to allow their component molecules to arrange themselves, and assume a state of more stable equilibrium—a state not so easily excited to the new combination.

This, which I offer more by way of analogy than

of direct argument, may fairly illustrate the condition of the blood, which has lately been in an unbalanced state as regards its constituents. I am far, however, from resting the truth of my general proposition on any arguments drawn from the symptoms of relapse, however these may seem to answer the conditions, because I conceive that further study may be well bestowed on the subject of relapses.

That form of rheumatism which has been called synovial, which was first distinguished from the fibrous by Dr. Chambers, and first made public in this very theatre, at its first opening by our learned registrar,* is a branch of our subject apparently encumbered with considerable difficulties. Its distinguishing symptom is the effusion of fluid in great abundance into the synovial cavity. The external features differ from those of the fibrous form, being not accompanied by that exquisite pain, nor by much redness or swelling of the surrounding tissues, although there is a similar enlargement of the joint. The urine is not so high coloured, and the extension of the disease to the heart does not so often take place. All these indicate a less acute form; and the absence of extension to the heart shows that the effusion into the synovial cavity has restored a degree of equilibrium, sufficient to save

* Dr. Hawkins' Gulstonian Lectures.

the extension to more vital parts. The relief afforded to the local vessels, and through them, in general, to the mass of blood, is, I believe, thus, as a general rule, preservative against extension; but when the effusion is of no great extent, or is absorbed, which is sometimes the case, then the vital organs are found to suffer. I avoid, in general, citing cases, as examples of the various forms are so familiar to every one; but I may, however, remark, that at this moment I have a case in the wards of St. Mary's Hospital: a youth of sixteen, who, when admitted, with effusion into the synovial capsule of the knee, had scarcely any redness of the surface, or pain on being touched. He had all the usual symptoms; the effusion disappeared in the knee, and the heart became affected both externally and internally. I regard this as an exception, the general rule being that effusion in sufficient quantity saves the extension.

I examined the blood in this case and found as follows:—

	In 1000 parts.
Water . . .	826·319
Fibrine . . .	6·838
Solid residue . .	173·747

Which shows a smaller proportion of fibrine than in the analyses given from acute cases by Andral and Gavarret.

Although in the two forms of rheumatism the

joints may be equally enlarged, their external form differs materially, depending, in the one case, on effusion within the capsule; in the other on diffused swelling and effusion in the cellular tissue surrounding the joint. The seat of inflammation is, I believe, different in the two cases. In the fibrous form I assume it to be situated in those vessels which I have demonstrated to be absolutely seated in the fibrous tissue proper; but in the synovial, I assume it to be situated in those which, in health, are employed in furnishing the blood from which the synovial fluid is formed. The contiguity of the vessels engaged in this last form of rheumatism to the internal synovial membrane facilitates their relief by means of effusion through the free surface of the inner lining of the capsule of the joint. This mode of relief does not lie so immediately within reach of the vessels of the outer fibrous tissue, which are those more immediately engaged in the fibrous form of rheumatism. One argument used by Henlé to oppose the general view of obstruction as a cause of rheumatism is this—that it is not easy to perceive in what way the removal, from the general mass of blood, of a quantity of serous fluid, should tend to restore the balance of the constituents, when the disturbance of that balance consists in excess of fibrine: he urges that it would appear likely to increase rather than to diminish the already too large proportion of the richer materials. To meet this

difficulty, we must bear in mind, that the fibrine and albumen of the blood, are employed in the nutrition of the body, and that the increase of synovial fluid (which is a true secretion consisting of water and albumen), must be formed, in some degree, from the nutritious materials of the blood; therefore, the formation of this fluid may as easily be the means of relieving the blood of a portion of its richer materials, as may the processes of nutrition in any other part of the organism. Thus, to a certain degree, it may abstract a portion of fibrine, or, more probably, it may abstract those constituents from which the fibrine is elaborated.

The fluid which is effused in the capsule of the joint collects insidiously, and its formation is not detected till it amounts to a considerable quantity. The nature of the fluid varies very much from that of pure serum, with a small quantity of albumen, to that of a more mixed fluid in which lymph to a variable extent is included. The varying composition of the fluid depends upon different grades of inflammation—that which approaches nearest to the natural character of the synovia results from the more subacute grade. The fluid when not containing much lymph seems to be readily absorbed, and usually is so on the subsidence of the local inflammation. Even when small portions of fibrine are contained in it, there is equal reason to believe, from recent observations given by Robin and Verdeil (to which I may afterwards refer), that

lymph which is poured out in effusions of this character may also be absorbed.

But that certain portions of fibrine may yet remain behind, and become organized, even in the synovial form of rheumatism, we must believe from the interference with the free movement of the joint, which generally remains in some degree after recovery; and this agrees with the general and very just opinion, that inflammation never occurs in any part of the system without leaving some trace behind. The damage is, however, of a graver character than mere interference with the movement of the joint, because, in case of a renewed attack, the power of relief is diminished; for there fluid is not again poured out. Equal relief cannot, therefore, be obtained in a second as in a first attack.

By this permanent alteration of structure in the joint, following an attack, to whatever extent it may exist, and however it may have taken place, we are led to understand why the localization of the rheumatic type of blood becomes constitutional, and we may deduce an auxiliary reason by which to answer the difficult question, which we discussed, just now, so much at length.

The extension of rheumatism beyond the joints now claims our consideration. The attack of the joints being the primary feature of the disease, the extension is no less characteristic, and is a frequent, though not a constant sequel.

If the local attack, consisting in obstruction,

subside before the general mass of blood is restored to its natural state, and the excess of fibrine remain unchanged, the obstruction may then proceed to the same tissues in the chest in which it has been localized in the joints, and the heart and its coverings may become affected. The pleura also occasionally succeeds in the involvement.

Now, if the heart had been the first seat attacked, it would not have been called rheumatism, until the joints had been affected; and if the joints should never have become involved in the localization of the disease, it would never have been called rheumatism at all: yet the nature of the seizure is the same, and the cause the same, although in the heart affection it would be classed under common inflammation. There are instances lately recorded of persons who have previously had the joint and heart affection together, being subsequently attacked by heart inflammation only, the disease being then simply considered pericarditis, in which I entirely agree; but it is evidently a part of the same constitutional affection, which had been called rheumatism in the first attack, when it occurred in conjunction with inflammation at the joints.

If we cannot wholly penetrate into the causes which govern the choice of localization, nor state why the over-fibrinated blood should sometimes become obstructed in vital organs, sometimes at the joints, we may, I think, be satisfied with repeating

our former remark, viz., that some structural formation, or mere accidental impairment of structure, or even functional derangement, may render a part more vulnerable, and so determine the localization thither.

The recently-recorded cases to which I have alluded bear out a further suggestion, viz., that though there may have been a greater liability in the first instance in the joints from their structure and offices to become the points of attack, yet that the permanent damage left behind in the heart, after it has been included in the first attack, has left that organ in a state still more vulnerable than the joints, when such a diseased state of blood again occurs to cause a second seizure. It is well known to practitioners that any pre-existing structural affection of the heart disposes to the complication of pericarditis in rheumatism.

The limits of these lectures only permit me to enter into the subject of the complications in a general point of view; and though I would gladly enlarge, I must confine myself to such considerations as immediately illustrate the views which I have taken of the general disease. The cause of effusions in the chest is identical with that which gives rise to the same effects in the fibrous tissues of the joints, but the results vary so far, that what is the general rule in the joints is the exception in the pericardium. In the joints, the general rule as to

the results is the effusion of serum or the increase of the natural lubricating fluid of the capsule. In the pericardium, the general rule is the effusion of organizable lymph, though serous fluid is often conjoined with it; the exception being that of more purely serous fluid. This difference in the effusion depends on different grades of inflammation, which in the heart arrives at a maximum in consequence of the impossibility of controlling the necessarily continued action of the organ. Were it equally impossible to obtain a state of rest for the joints, I believe the inflammation would reach an equal height in them. The tissues being of the same character, the distribution of vessels is the same, and the obstruction is the same, but there is not the same degree of condensation of structure in the sero-fibrous tissues of the heart as in the joints, and, consequently, there is not the same amount of liability to obstruction in the vessels of the membrane.

We deduce from this a reason why the chest affections stand as complications and not as primary seats of inflammation. The great irritability of the heart, as compared with other vital organs, places it next to the joints in liability to seizure, and before the pleura. The action of the heart is eminently uncontrollable.

The structural formation of the pleura differs from that of the pericardium, and the effusion varies

correspondingly: there is a less abundant supply of fibrous tissue, and the effusion is, in general, more of a serous character. Besides this structural reason accounting for the still more infrequent complication of the pleura than the pericardium, we may recognise another in the action of the lungs being more under control than that of the heart, and their motion being capable of modification, so as to give partial rest at the first tendency to obstruction. The internal membrane of the heart becomes also affected in rheumatism; and here again I should ascribe the cause to the intermixture of fibrous tissue in the parts. The most frequent seat is the valvular structure. The auriculo-ventricular valves are those most selected: the mitral by far more frequently than the tricuspid. I cannot wish a stronger testimony than this to my view, that the obstruction first occurs in the fibrous tissue, for the order of priority of attack in these valves is in exact parallel with the amount of fibrous tissue included in their structure; so coinciding with the rule which I have endeavoured to trace throughout all the organs. A reason for the greater liability of the mitral valve, has been given, as I think, with great judgment and accuracy by Dr. Law, of Dublin; he refers it wholly to the larger proportion of the fibrous tissue, and to this I would add that this valve has also a severer duty to perform than the other valves, viz., to oppose the whole force of the left ventricle in its

systole, and thus the liability to derangement, by the onerous nature of its function, combines with its natural organization to induce the selection.

Dr. Watson has shown in his beautiful and very ingenious description of the semi-lunar valves of the aorta, that when fibrine is effused on their free surface, it is confined to that part of each valve where the fibrous tissue* is distributed, and that those portions of the valve which overwrap each other are not the seat of exudation. He remarks on the possibility of the contiguity of the two surfaces of adjoining valves being a reason for the absence of lymph; a conjecture which does not seem to me quite so satisfactory as the general governing law of the existence of fibrous tissue.

Several distinguished authorities in pathological physiology (Gulliver, Simon, Kirkes, Lee, &c.) have observed on the mechanical detachment of fibrine during the circulation, and on its adherence to opposing parts or obstacles in particular states of disease.

Chorea is not a frequent accompaniment of pericardial rheumatism, but it occurs too often to be regarded in other light than as a complication. The subject is very difficult: Dr. Bright has been the first to notice it, but has not shown any patho-

* With the exception of the small bit of fibrous tissue which composes the corpus aurantii, and the thread of fibre which borders the valve.

logical connexion between rheumatism and chorea, and I do not hope for much greater success. We know chorea, in general, to be a disease of debility, and that the treatment by direct tonics, the effect of which is to increase the blood corpuscles, is always successful: we know that in rheumatism, there is, together with the excess of fibrine, an absolute diminution of the blood corpuscles; and that when the normal proportion of fibrine is restored, which takes place simultaneously with the disappearance of the active febrile symptoms, the deficiency of blood corpuscles is the only remaining defect in the constituents of the blood; a state similar to that of anæmia.

In regard to one of the prominent symptoms in chorea, the excessive excretion of urea, this differs but little from the excessive secretion of uric acid in rheumatism; uric acid being itself a urea compound, which would easily be converted in the organism into urea and carbonic acid by a small appropriation of oxygen and the elements of water.

Thus the symbol for uric acid is $C_{10}\ H_4\ N_4\ O_6$

Add 6 atoms of oxygen O_6

4 equivalents of water $H_4\ O_4$

We have $C_{10}\ H_8\ N_4\ O_{16} = 2\ (C_2\ H_4\ N_2\ O_2) + 6\ C\ O_2$, or two equivalents of urea + 6 equivalents of carbonic acid.

Or in an equation,

$C_{10}\ H_4\ N_4\ O_6 + 4\ H\ O + 6\ O = 2\ (C_2\ H_4\ N_2\ O_2) + 6\ (C\ O_2)$.

The difficult and important subject of local rheumatism contains so much of interest that a whole lecture would scarce suffice to do it justice. I must now dismiss it with little better than a slight allusion, and the more willingly so, because our proper subject is Acute Articular Rheumatism. It might, however, be urged by objectors to the principles I have advanced, that if true, they ought also to be applicable to the local form and meet all its attendant conditions, such application not being apparent, at least upon a cursory examination.

I believe that closer study would show the identity of the nature of the two forms, and refer them both to the same cause: the structure of the tissue involved is the same in both; but in the local form the seat is in the fibrous covering of the muscles instead of the fibrous coverings of the joints. The point which requires deepest investigation is an apparent difficulty in the particular localization. Certain circumstances of especial and partial exposure under, as I conceive, a necessary condition of the atmosphere, causing a local stasis, by constriction of the extreme vessels of the part exposed, are the leading points to be dwelt upon as guides to the solution of the difficulty: many considerations, including that of the previous state of the blood of persons so attacked, would require to be carefully gone into.

I hope to deduce further evidence in favour of the justice of the views which I advocate, from a com-

parison of the action of remedies, with the results to be expected, if that explanation of the rheumatic type of inflammation were admitted. But before tracing that line of testimony, I will briefly advert to the insufficiency of all those theories, already current, to meet the difficulties of the subject; therefrom not only deriving an excuse for a new suggestion, but showing an absolute necessity for fresh investigation. I wish, however, while reviewing other propositions, to bear in mind, that to undermine another's theory or proposition, does not, of itself, substantiate mine.

Various ideas of the nature and cause of rheumatism have obtained a certain degree of currency, and are received, even at present, as so many systems of explanation. For the most part they either embrace only superficial phenomena, or else assume for causes mere shadowy names, not understood as realities; it is impossible, therefore, that they should carry any satisfactory demonstration. In fact, I may add, that although they are allowed to pass current, I do not believe that they satisfy the mind of any inquirer.

The chemical basis of argument, drawn from the analytical examination of the secretions, from which the theory of "lactic acid" has been derived, lends to that result a claim upon our respectful consideration. For chemical analysis is a purely philosophical mode of pursuing inquiry; and the supposition

of the existence of some particular acid pervading the system is at least plausible, when rested on the obviously acid state of the secretions.

Assuming, for a moment, the existence of lactic acid in the system, we have not, by doing so, advanced a step towards establishing a cause for the local affection of the joints, or for the so-called "metastasis." Supposing that the lactic acid should be present in the system, it might be found as an acid in the urine and in the perspiration; but in the blood, which is always essentially alkaline, it could exist only as a lactate; and it is to the blood alone that we can look as the agent to convey this "morbid poison" to the joint, and to determine the affection there. I have myself tried the reaction of blood drawn during the presence of most acute symptoms, and have always found the serum alkaline. An intricate process of elaboration might, by an effort of imagination, be conjectured in the joint, to account for the localization; but the data for such a supposition are, at any rate, too far beyond our reach to warrant the construction of any hypothesis in this manner. I conceive, moreover, that further research in this direction would be quite unprofitable, because the actual fact of the existence of lactic acid in any of the secretions has been strongly controverted, if not absolutely disproved. We have the testimony of Liebig, Enderlin, and Pelouze, that they have been unable to detect any such acid in the secretions.

The experiments of Liebig were made on an extensive scale, under all conditions of the urine. I quote his words:—"It is precisely in analysis of the blood and urine of man and of carnivorous animals that we find lactates mentioned as constant constituents, not because they have in reality been detected in those fluids, for no one has as yet succeeded in producing lactic acid therefrom, but because in examining the aqueous and alcoholic extracts of blood and urine, some non-crystalline matters have been found which sometimes manifested an acid reaction, and upon incineration left a carbonated alkali as a residue; thus presenting a similarity in deportment to the alkaline lactates."* This shows that the existence of the lactic acid has been merely assumed by a sort of system of exclusion. Surely an inference founded only on a guess must be regarded as a weak basis for pathological reasoning. But I quote again still more to the point, from the same great authority: after reasoning upon the probable absence of lactic acid from the nature of the food of carnivorous animals, he proceeds to say: "The assumption, *à priori*, that neither the blood nor any other fluid of the body of carnivorous animals can possibly contain any lactic acid has been positively established by the experiments of Enderlin. Finally, Pelouze has proved that the experiments of Henry, who pre-

* Simon's Chemistry.

tended that he had detected lactate of urea in urine, are erroneous, and by no means to be relied on." *

We have as yet derived from chemical research no satisfactory explanation as to the acid reaction of the secretions. Although we know that the acid in the perspiration is volatile, yet its exact nature is very doubtful; and I shall dispose of this difficulty of accounting for its presence as a special symptom of acute articular rheumatism, by denying its existence *exclusively* in that disease. I have already stated that it is common to all attacks of inflammatory character, and more particularly to such as are accompanied by pain.

I have yet to give you the remarks of a great German authority on this topic:—Henlé says: "Among the theorizers who have felt the necessity of seeking more narrowly for the rheumatic principle in the nature and peculiarities of the blood, some have been led by the sour smell of the perspiration, the acid reaction of serum from blisters, and the quantity of uric acid in the urine, to suppose that the blood is overcharged with an acid, which

* These experiments have been performed by a very clever chemist, Mr. Medlock, in which I have assisted; they were repeated on 40 lbs. of urine. A considerable quantity of a zinc salt of a non-volatile organic acid was obtained. The experiments are still in progress, by which it is not only hoped to obtain the nature of the acid contained in putrid urine, but also of that to which fresh urine owes its acidity.

they conceive to be lactic acid. I will not dwell on the objection, that the very existence of this acid in the system generally, and the manner in which it would be retained in the blood in particular, are both problematical; nor, that it is difficult to understand how it is conveyed to the joints and heart, and how it may excite any especial form of disease in those organs. We must give and take permission to be mutually lenient upon many unguessed riddles of this kind; but the hypothesis is as objectionable as many other dilettanti mystical-medical suppositions (mystische - medicalische), which only attempt to hang together a couple of ideas, without displaying in what way they may be attached: it is objectionable, because the connexion in which the acid and the symptoms of acute rheumatism really stand to each other is the very reverse of that which the theory requires: there is no reason to suppose an accumulation of acid in the blood, for the acid excretions which appear in larger proportions during the disease than in health are the very reverse of a proof that there is an accumulation of acid in the blood."*

So far Henlé. Had I myself ventured on such a tone of animadversion, I might have been suspected of being disrespectfully facetious; but given as a literal translation of the words of a profound

* Henlé, vol. ii.; Handbuch, p. 266.

and grave German professor, it need not be suppressed.

Leaving, then, the hypothesis founded on the gratuitous assumption of "lactic acid," we may still find many speculators who, without including any material so definite, treat of the original cause of rheumatism under various designations: such as "morbid influence," "rheumatic poison," "materies morbi," "diathesis," "mal-assimilation," &c., while others inclining to the "lactic acid," but not being very sure of their foundation, add one or more of these expressions to strengthen their position. Terms, however, which are mere arbitrary substitutes for knowledge, need hardly occupy our consideration.

Although the process of sudden relief, and the so-called "metastasis" have not, that I am aware of, been explained, yet many reasons for the localization have been brought forward. The most recent is that of exalted nutrition of the part by which the locality is said to be enabled to attract more of the "morbid poison" to itself. It is meant by this, I presume, that a more exalted state of nutrition enforces or attracts a larger supply of blood to the part, which blood is contaminated by the unknown poison. Supposing this to be the case, unless the part should also possess the property of neutralizing that poison, there is no obvious mode of accounting for the departure; and if we include the existence of such a power, we find three purely

hypothetic assumptions in one short argument, which can hardly be called a very luminous mode of explanation. The three are, of course, the exalted state of nutrition,—the poison in the blood,—and the power of neutralizing it.

If we refer once more to the pages of Paulus Ægineta, we shall find that, holding something of the same idea, he has been wiser in his generation. Being conscious that the continually-present weakness (a term which in him corresponds with exalted nutrition) cannot consist with the sudden relief—" for the paroxysm then should be unintermitting"—he makes friends with another adjuvant, flies to the peccant humour, and associates it, as an equal cause, with the continual weakness. He does not, however, strengthen his position by the alliance.

Nervous influence is a word which appears to have an approach towards a meaning. It has been especially framed to meet that mysterious characteristic, the sudden shifting of the points of attack. Reasoning from the fact that in the spinal marrow all the nerves of the body unite, it is inferred that through it, as a sort of centre of communication, any locality may be attacked. Henlé has handled this topic by ridiculing the idea, that having traced the disease to the spinal marrow, we shall hence borrow an excuse for imagining a migratory power: " Why," he says, " should we take it for granted that it may wander up and down?" He continues his refutation

by saying, "that when nervous pains do migrate, the change of seat is not arbitrary, but according to the laws of nervous sympathy. The organs to and from which acute rheumatism makes its 'metastasis' are not selected according to these laws."

Among the indefinite terms adopted, in order to supply the absence of clear ideas on the nature of rheumatism, none are more unsatisfactory than those of predisposition and hereditary influence.

The word predisposition in itself conveys nothing explanatory, and we should not throw much light, if for diathesis we were to substitute a certain constitutional tendency toward the production of lactic acid, &c., nor even, as I would rather express it, towards a particular susceptibility of the organism to be acted on by atmospheric causes, which result in an over-fibrinated condition of the blood. Nor do we advance much further by endeavouring to trace the transmission of such constitutional liability to the parent, and thus set up the vague hypothesis of hereditary influence. That rheumatism may in a general sense be inherited, is a fact beyond all controversy—because, together with all other diseases, it must be transmitted with every other condition of humanity from one generation to another. It is also a self-evident truth, that there being more persons than there are diseases in the world, a recurrence of any one disease, especially if it be a common one, must frequently take place in any one race or

family, and the number of recurrences in all families cannot be equal.

The two propositions, therefore, that a general constitutional tendency to all diseases is transmitted from one generation to another, and that some diseases must appear oftener transmitted than others, deserves neither affirmation nor denial, for they are self-evident truths which require no statistics to support them.

That there is a proneness in some individuals, at any one time, to be more affected than others by external causes, is certain, because many may be placed in the same circumstances, and only one attacked. The hereditary question consists in this, whether that greater degree of proneness has been the result of the existing state of the system, produced by a series of combinations of external circumstances, acting on the organism, or whether it has been actually born with the individual constitution? We have certainly the palpable fact before us, which may be verified by daily observation, that personal characteristics are to be traced in descent; and analogy would lead us to decide from this, that not only may the liability to disease in general be handed down, but that a particular state of organization, obnoxious to certain external influences, may also be transmitted. But if we assume this as demonstrated, and accept hereditary influence in its particular as well as in its obviously general sense,

we shall still only refer that particular type of organization from the present to the first sufferer, and so be able to affirm that the first man being predisposed, a certain number of his descendants became therefore predisposed: we should thus only become possessed of the word predisposition, as describing the state of an individual, instead of any rational account of what that state of organization consists in which we are investigating.

I cannot say that I regard the slight attempt which has hitherto been made at statistical inquiry, in regard to the hereditary character of rheumatism, as at all conclusive. It would require a most laborious and widely-extended statistical return to prove that this or any other disease obtains more frequently in any one line of descent than in another; and the intricacy of such inquiries, arising from the intermixture of blood, by the double parentage, would render such an investigation all but impossible.

The most attainable and useful result of statistics would be the comparison of the frequency of recurrence of diseases among classes, subdividing the labouring portion into their several trades or employments. The prevalence of disease in different classes, viewed with reference to habits, would afford scope for many valuable inductions; and it is to be wished, that the industry with which most inquiries are now prosecuted might be directed to this point.

If it should appear, for instance, as I have no doubt it would, that rheumatism prevails especially in classes in which labour and exposure to atmospheric causes are the conditions of the state of life, the prevalence of the disease among them would be more easily referred to the fact, that the same calling had been often followed by successive generations, than to the indefinite idea of any especial hereditary disposition.

LECTURE III.

In my last Lecture, after recapitulating the physiological data, and sketching the argument which I deduced from them, I proceeded to examine the symptoms which especially characterize rheumatism, and applied to each that principle of explanation which had been the result of the argument. I discussed more minutely the view proposed in the first lecture, both of "obstruction" and "metastasis," considering a solution of fibrine in the joints to be the regular consequence of the stasis, and of the absence of further supply of oxygen to the blood obstructed. The probable reasons for localization were examined, and that circumstance dwelt on as indicating the difference between common inflammation and the rheumatic type.

The general localizing cause was referred to the structure of the organs usually affected; and the choice of individual seats of attack, attributed either

to some peculiar formation, or to damage or impairment of the part, either by undue use or by accidents. In cases of recurrence, I referred it to the lesion remaining after a primary seizure.

I specified the usual order in which the joints were attacked, with the reasons which seemed to account for the preference. The greater liability of children to be attacked by rheumatism and its complications were referred to their larger relative proportion of fibrine in the blood, and to their greater irritability offering less opportunity for reparative repose.

In applying the same general principles to synovial rheumatism, the vessels of the immediate capsule were regarded as the seat, in contradistinction to those of the fibrous tissue proper, as I would term it, which are the seat in fibrous rheumatism; the gorged state of the vessels equally constituting a stasis, and the heart's force equally acting to induce effusion, and the same sort of self-relief to the vessels equally following; the effusion into the capsule, being the more readily induced as being a natural secretion.

The result of the process I supposed to be of the same generally restorative tendency to the whole mass of the blood. A foundation for habitual obstruction, leading to chronic rheumatism, appeared as the consequence of certain slight permanent damage remaining in the joints after effusion. I

then passed to the extension of rheumatic inflammation in the complications, in which the identity of the tissues implicated mark the course of the extension; and, still referring the first cause to general inflammation, I begged you to consider that, had the complication preceded the affection of the joints, the attack would have been regarded as simply pericarditis, and would have been finally so classed in case the joints had never been involved.

In effusion in the chest, the same relief to the general mass of blood is to be attributed to the process; but danger is to be feared from the peculiar nature of the locality and the construction and offices of the organ involved, the danger being more future than immediate, since the adjustment by absorption in the temporary cure is the prevailing rule.

On the subject of chorea, the want of obvious connexion between the two diseases is but very partially supplied by tracing a similarity in the nature of the blood, and by reasons which imply the probable existence of urea in excess in the urine both in the sequel of rheumatism and in chorea.

The various theories which are most in vogue were then passingly adverted to; and though the award of being the most philosophical might be accorded to lactic acid, it was found to be wholly untenable for want of actual data on which to plant a surmise. The less substantial hypotheses of morbid influence, diathesis, poison in the blood, and exalted

nutrition, &c. &c., being mere expressions instead of tangible propositions, did not detain us long. The idea of nervous influence proved also, to my mind, altogether inadequate as an explanation. To a certain extent I urged the acceptance of hereditary influence in the light of a self-evident proposition, but one which, though in itself unassailable, was not more applicable to rheumatic disease than to any other condition of the human frame. Admitting the fact according to its fullest meaning, it was merely referring the constitutional effect, whether of rheumatism or of any other disease, to a remoter generation, without at all explaining its nature. I ventured to suggest that the occupations and modes of life of the persons amongst whom the disease chiefly prevails would be the most valuable direction in which to prosecute statistical inquiry.

While canvassing former opinions, I must once more observe that I am perfectly aware that to demonstrate the failure of other hypotheses is no argument in support of that which I wish to advocate. That other guesses should be wrong cannot, by any perversion of logic, prove this one to be right. The chief use of examining and rejecting is to disencumber the subject of certain indefinite ideas, which, by passing current for more than their real value, are apt to satisfy the mind and make it apathetic in the pursuit of closer inquiry. Even in this enlightened, inquiring age, it is amazing to observe the multipli-

city of terms in constant use which are only substitutes for thought and reason.

I have always considered that dissertations on treatment require an apology when, in this place, they are brought before the most accomplished practitioners of England. To dilate on general well-known modes of practice is surely out of place; to offer the lecturer's individual experience is anything but respectful, unless the disease under consideration be rare, or be reviewed in relation to some newly-proposed principles. Having, however, entered on a train of explanatory argument, we must not neglect any evidence by which its soundness may be tried: for even if our proposition, in its present state, be regarded as merely another of those innumerable guesses at truth, it must obviously stand or fall according to the number of conditions which are answered by it. If a comparison of the state of the system and of the alleged causes of the disease be made with the known working of various remedies, corroboration or doubt may be thrown on the correctness of those assumed causes; and if, reasoning from those causes, we find it possible to assign satisfactory explanations for the action of very opposite remedies, we shall obtain strong testimony to the exactness of the principles which we would establish.

It is not wonderful that, since such indefinite ideas have been held on the pathology of rheumatism, the modes of treatment adopted by practitioners should

be varied and incongruous in the extreme, and that they should rise but little above the character of merely tentative applications. Accordingly the most discrepant classes of remedies have been in favour, and a certain degree of benefit has accrued from nearly all. We have had bleeding and active purgation; we have also had bark, with opiates; we have had acids given furiously, and alkalis to saturation; salts, sudatories, and endless specifics; and all of these more or less successful in the hands of men of such character and ability, that it is not possible to doubt the certain degree of efficacy which has been attributed to each. As these various modes of practice, followed in a manner empirically, because under acknowledged doubt of the nature of the cause to be attacked, have, nevertheless, given a certain degree of general satisfaction, they may afford us a useful test of the truth of our argument. We may compare the way in which the remedies may be conceived to operate with the principles which we suggest for the causality of the disease; and if reasonable explanation is afforded for all these results, which seem contradictory to a superficial view, we shall have strong corroborative evidence of the soundness of our hypothesis. To review, also, the nature of the action of so many discordant modes of treatment will naturally lead to the choice of that which is most logical, and, therefore, most likely to be successful.

Of all practices in medicine, bleeding has been most capriciously adopted. None is more valuable, none more dangerous. In some cases it has been carried to terrific extent; in a vast number wholly neglected, when its application might not only have met the existing symptoms, but have saved a long series of supervening evils. The reason to be assigned for this is, that venesection has been hitherto adopted to meet symptoms which are patent to the eye; and as it is now known that symptoms which are similar may arise from very dissimilar causes, and during different states of the blood and secretions, it is not to be wondered at that grave mistakes and irreparable failures have often been apparent, and that the practice has fallen into disfavour. The best of us have at times pursued it, guided by no more certain light than a practical knowledge of the circumstances under which it may be safely trusted; and I believe in the use of this, as well as of other remedies, all have felt, with pain, that they have had to substitute skill for science.

But perhaps rheumatism, under the assumption that the views which we have held out are correct, affords the best-known instance of a disease in which bleeding might be applied or withheld on solid principles, that is, in which something of an exact knowledge might be obtained of the nature of the effects produced by depletion, so as to judge of the time when

it should be used, and the extent to which it might be discreetly carried.

In rheumatism, bleeding has been used to a fearful extent, with a view to cut short the attack, or, in other words, to cure the disease; and Bouilleaud extends his confidence in its efficacy still further, for he states that large and repeated bleedings guard the system from those complications which result when the disease extends to the chest. Many testify to the success of the practice, though I have long considered it dangerous and inexpedient; and I have no doubt that many agree with me. Examination of the cases of Bouilleaud also disproves his assertion that they have been less subject to subsequent complications than usual.

Now if it can be made apparent, by examining the state of the blood after successive bleedings, that venesection does not in any case reduce the relative proportion of fibrine in the blood, but that, up to a certain period, the excess of fibrine goes on increasing, we obtain sight of a reason why the practice should not be successful in any radical manner; for no amount of abstraction of blood can remove that unbalanced condition of the constituents which I look upon as the essential character of the disease. The fact that the relative proportion of fibrine is not altered by the abstraction of blood is shown by the Tables given in page 52.

The question then follows, that if bleeding cannot act beneficially by restoring the balance of the faulty condition of the constituents, in what way can that remedy have acted when its application has been followed by a certain amount of temporary relief, of which many have borne evidence, and the truth of which, in a modified sense, I am far from wishing to deny?

A direct relief afforded to the pain in the joints may be referred to the consequent lessening of the heart's force; for whenever there is a stasis of the blood (and we have assumed, in the case of rheumatism, such a stasis does take place in the vessels of the fibrous tissues of the joints), then the impulse of the heart, forcing a further supply on the already gorged vessels, communicates a renewed pressure at every beat, occasioning most violent pain. A portion of blood taken from the system, by lessening the whole quantity in the circulation, must relieve the pressure. A single bleeding, therefore, during excessive pain in the joints, is a practice for which we can assign sufficient reason. The quantity should be moderate, and the act of venesection not repeated, for reasons which I shall now further state.

Another change in the blood, made apparent by this evidence of the Tables (page 52), is a diminution of the amount of blood-corpuscles after each successive bleeding, as well as an increase in the quantity of water, both of these changes marking a condition of

the blood approaching to that in anæmia; for since, notwithstanding the bleeding, the relative proportion of fibrine goes on increasing, and the amount of corpuscles diminishing, the strength of the patient (being directly as the number of corpuscles) must be dangerously reduced by repeated losses of blood. The fibrine, it appears, still draws on the corpuscles, the latter having to be supplied from the proper nutriment of the body. The Tables (page 52) show that the excess of fibrine goes on increasing up to a culminating point, and then recedes. The first bleeding, which we have already described as more a palliative than a curative measure, may, however, act beneficially by removing some of the material of which fibrine is formed, viz., the blood of corpuscles, and thus accelerate the arrival at that maximum point. We may also conclude that, if the certain maximum period could be precisely ascertained, blood taken at that particular crisis, in proportion to the degree of strength remaining in the patient, would abstract not only the largest relative but the largest absolute amount of fibrine, and thus abate the cause at the least expense.

The advantage to be gained by loss of blood at this crisis might be further illustrated by reviewing the different effects produced on fibrine by different degrees of inflammation. This topic, however, we must defer for the present, while we say a few words on the action of local depletion, for the benefit de-

rived from which we may assign sufficient reason to advocate its more frequent adoption than has been the practice with many good authorities. Local bleeding not only relieves the distended vessels of the surface, and indirectly those which are deeper seated, but it also abstracts a larger proportion of fibrine than can be effected by taking the same quantity of blood generally.

The fact is fully recognised that blood taken from the surface capillaries, whether by cupping or by leeches, contains a larger proportion of fibrine than that which circulates in the larger vessels, *i. e.*, that surface blood is richer in solid or coagulable constituents than either venous or arterial blood. The proportions given by Dr. Pallas* are 3·10 : 2·55, and 2·63 : 2·55. This is denied by Denis,† but his analysis of blood taken from the same individual, from the arm and by cupping, bear out the same results.

This commendable practice has been superseded in many cases by fomentations, which have also a useful tendency, inasmuch as they forward the transmission through the capillaries of the blood, too rich in fibrine, by diluting it with the fluid used. It is probable that this effect is produced by the physical act of endosmosis, yet there is no reason why

---

* Journal de Chimie, Simon, vol. i., p. 217.

† Recherches, p. 72.

fomentations should not be combined with local bleeding as a double method of relief.

A very similar result to that of venesection is obtained by the continued action of purgatives, and is effected in a more legitimate, that is, natural manner. Could the circumstances of the patient afford of their being freely used, they would be eminently useful. In purgation the effect of the remedy causes abundant secretion from the lining membrane of the canal, and by that secretion the blood is relieved of large portions of its richer constituents, as well as of its water, and the blood is freed of albumen and fibrine without suffering any diminution of the blood corpuscules. In this last important fact consists the essential advantage to be gained by this mode of depletion over that of absolute abstraction of blood. It is to be regretted that a great impediment to its use in rheumatism is felt in the increase of immediate suffering produced by that movement of the fixed and exquisitely painful joints, which is inevitable during the action of the medicine. Although, perhaps, practitioners have not been guided to this mode of relief by reasoning on the saving of red corpuscles as its especial claim for preference, yet the good result has been so practically apparent that few neglect to use it, at the least to assist in the progress of recovery when the intensity of pain is subsiding.

The action by endosmosis has been contravened ;

but although action which may be otherwise accounted for has been incorrectly attributed to endosmosis, yet no argument has been produced which, to my mind, invalidates the received opinion, that saline fluids act endosmosically according to some law of their density. The point does not lie near enough my subject to induce me to digress to argue it, especially as I must leave several questions untouched which I should gladly have discussed had time permitted.

The constitutional effect of mercury as it is generally understood, *i. e.*, distinctly as to its result, but vaguely as to its mode of working, will not throw much light on the subject we are especially discussing. Its acknowledged use is to controlthe exudation of lymph. The precise mode of action of mercury upon the blood remains in obscurity; but we know that it must have some specific effect on the constituents of the blood, because it checks the exudation of fibrine, and it must act in some way to modify those constituents so as to restore the balance.

The stasis which takes place in the fibrous tissues of the joints during the local affection, being the result of an earlier grade of inflammation, does not often result in the exudation of lymph. At this period mercury is very seldom given, probably because experience has not testified to much advantage derived; and as we thus perceive that the only action

which we can attribute to mercury (being to check exudation) is not required at that period, we have a sufficient reason to account for the practical result.

Supposing, further, that mercury possesses the power to which we have just alluded, of directly influencing the constitution of the blood, although we might then regard it as a means of working beneficially on the general mass, yet we should not anticipate an effect to any sensible degree in the local affection of the joints. That small quantity of blood which has become retarded in the joints, remaining there stationary, and, as it were, out of the circulation, would be occupied in doing its own duty of obtaining self-relief through the solution of the fibrine, and being less affected by what takes place in the blood generally, would partake but very little of the effect of mercury in the entire mass. All this falls in with the result of practical observation, which has led to the disuse of mercury, in order to attain the constitutional effect during the local attack. The period when it is used is the extension of the disease to the chest, and the effect of the remedy to check exudation is too fully appreciated to require much further comment. The effect of the more intense grade of inflammation is to produce some change in the constitution of the fibrine, which either enables the vessels to pour it out, or enables the fluid to permeate their coats and exude itself on the free surface of the membrane. When

the disease reaches the vital organs, the inflammation is of this intense character. It appears to me that the constant action of the heart is one of the causes which induces this higher state, and that if the joints were perpetually moved during their attack, the exacerbation there would amount to something of the same degree.

The necessity to check exudation, when this high inflammatory state exists in the shut cavities and organs of the chest, arises from the peculiar locality; for though we should desire to see fibrine pass off, in order to give general relief to the constitution, still its presence in those closed localities is of more dangerous consequence than even in the general circulation.

Amongst many practitioners even this mercurial treatment is beginning to be discontinued, because an impression is obtaining that the disease can be cured without it; yet I should advocate its use, because the remedy, through this check of exudation, carries the disease to a favourable issue with a smaller amount of remaining mischief. Although it may be true that, as a general rule, patients surmount the acute attack, yet most of them become subject to severe subsequent complications, under which the constitution ultimately sinks: the use of mercury is to lessen the degree of foundation laid for all these future complications.

Pursuing, as we proposed, a review of the various

opposite modes of practice which have been found of use, and the extent to which their success may be accounted for agreeably to our proposition, we will now consider the action of tonics and sedatives.

There are testimonies of high respectability, that bark has been used with success in the treatment of rheumatism, from the very beginning of the disease, and at a time when the pulse has reached a hundred in a minute.

Statements to this effect have been accompanied with the remark, that the blood when drawn has always exhibited the buffy coat.

It is impossible to impugn the truth of such authors as are cited, but difficult to understand the statement in which a contradiction is implied.

The description of the state of the blood drawn is a proof that in those cases depletion has been used as well as tonics; and the fact of depletion having been used, affords evidence against the previously-stated position that tonics have cured the disease from the beginning.

Modern chemistry has taught us to appreciate the value of bark in medicine by a comparison of the organic bases of vegetable substances. There is a certain number of vegetable substances from which the essential principle or alkaloid has been extracted. These alkaloids, when compared in their elementary constitution, are all found to contain nitrogen, and, according to their proportion of carbon and nitrogen,

they form a series, of which the poisonous alkaloid strychnia stands at the one extreme, and the harmless caffeine and theine at the other: while the medicinal alkaloids, quinine and morphia, occupy a middle place. They are all stimulants of the nervous system, and all more or less promote the vital functions. It is easy to suppose that this promotion of vital energy may be overdone, and so carried to a destructive extent by the extreme and poisonous stimulants, strychnia for example; and it is also easy to suppose that the medium, but still very active, stimulants, quinine and opium for example, may be assistant in some states of the nervous system, over-active and deleterious in others. The palpable evidence of the action of the extreme alkaloid is seen in the tetanic convulsions produced by strychnia.* Nor will it be less obvious that the action of the medium medicinal alkaloids is sometimes good and sometimes injurious, if we contrast the improvement of the processes which terminate in nutrition produced by quinine, and the production of febrile symptoms by the same medicine.

Bark, therefore, we see, in promoting the vital functions, may act to invigorate those peripheral processes which are in defect, and to reinduce the employment for purposes of nutrition of that fibrine

* Strychnia, though acting on the whole nervous system, shows itself more especially in disturbance of the functions of the cerebro-spinal system.

which exists in excess in the blood, and so help to dispose of the fibrine, and restore to the blood a balance of the constituents.

This would be acting at the opposite end of the machine. We have hitherto only considered the mode of disposing of the fibrine in excess by removing it absolutely from the circulation with a certain mass of blood; but we now suppose it probable that, by giving the stimulant alkaloid, a similar effect may be produced in the blood by increasing the nervous energy at the periphery, and thus inducing the elaboration of the fibrine into the system, to replace the effete tissues of the organism.

It may be objected that typhoid fever, in which bark is known to succeed, is a very opposite state to that which we have been considering with an over-fibrinated state of the blood, inasmuch as there is a deficiency of fibrine in typhoid fever. We can only suggest that the exalted condition of the nervous system, which is induced by the stimulant alkaloid, may only be corrective of error, and act to restore the healthy condition whatever that error may have consisted in; whether it be in defect or in excess of fibrine in the blood.

Liebig has advanced an idea that quinine assists in the formation of the brain itself, by supplying elementary constituents to replace those which are become effete, and thus through direct reproduction the whole nervous system is excited to new and

energetic action. This, if true, leads directly to support the view that the peripheral action (by which the fibrine is employed in the nutrition of the body) is increased and excited through the stimulating effects of quinine. It supports equally the supposition that whatever be the process in defect, that process will be forwarded by the remedy, and act curatively, as far as nutrition can have a remedial tendency.

The fact is, the beneficial action of bark is, in the early stage of rheumatism, most questionable: my own opinion is that it is only when bleeding, used with judgment, has prepared the way, or when the maximum point is passed, that any real advantage can be derived, while a certain degree of doubt or actual danger attends its earlier use.

Though the alkaloid colchicia has never yet been analyzed, colchicum is known to be one of those poisonous vegetables from which these essential principles are extracted. It has been too favourite a remedy to permit us to omit to notice it, nor can we deny that it has the property of acting directly upon the diseased state of the blood.

The degree of benefit which has been derived from it is a fact coincident with our view of the disease, for, acting as a violent irritant of the mucous membrane, it draws from the blood, in the form of secretion, its richer constituents.

The effects, however, are so variable in different

constitutions, and its properties so diverse in different preparations, depending on casual circumstances, that its action lies too much beyond the power of calculation to make it a very safe or desirable remedy.

I am disposed to attribute some of the sudden cures ascribed to colchicum to the circumstance that the local affection usually disappears after a brief space, with or without the application of any remedy, by the self-relieving process, which has been the foundation of nearly all our remarks. This relief, which is certain to happen, is almost certain, from its suddenness and completeness, to be attributed, by the patient at least, to the last-taken medicine.

With a view to control the effects, so as to secure a moderate and less alarming action, it has been usual to unite colchicum with alkalis or alkaline salts. These are said,* however, to precipitate the alkaloid, and, by rendering it insoluble, probably to make it altogether inert. This, if true, is one of the numerous instances which admonish us to be mindful of the paramount importance of sound chemical knowledge in the art of prescribing remedies. It is grievous to reflect on the facility with which incompatible substances may be associated; the least though still serious consequences of which must be to miss the aim intended.

---

* Brande.

Opium, from which various alkaloids have been extracted, is not imagined to have any specific effect on rheumatism. Since our purpose in examining the action of remedies is to illustrate the nature of the disease by their effects upon the blood, opium will not assist our subject. Its anodyne power renders it a useful palliative, by diminishing sensibility and muscular contraction: it is also known to check the secretions, excepting that of the skin.

Much has been said on the use of alkalis and acids in rheumatism. The former have been given from the acid nature of the secretions; the latter upon a rather more philosophical principle than this, because the salts of vegetable acids pass through the organism as alkaline carbonates: but this is suggested merely by the observed state of the urine, and not with any reference to the general state of the system. The alkaline treatment being especially directed on the results of the disease, viz., the acid state of the saliva, the perspiration, and the urine, does not attempt to reach the cause, but only the effects: and the blood being essentially at all times alkaline, and being at the same time the medium through which all the characteristics of the disease, the inflammatory state, the fever, the affection of the joints, &c., are induced, an attempt merely to saturate the system, as it is called, by alkalis, must be ineffectual as a curative measure.

A better-founded reason for the use of alkalis and

alkaline salts may be deduced from the consideration of their power as oxidising agents, by which power the water of the blood may be decomposed, the oxygen going to oxidise the uric acid, and the hydrogen to form ammonia.

The acid nature of the secretions, a state not so much in opposition to that of health as in excess of it, even in rheumatism, exists to so small an extent, that though the urine can be obtained in abundance for experiments, the very nature of the acid has not yet been ascertained. In consequence of the supposed volatile nature of the acid, the urine has been distilled, and hydro-chloric acid has been obtained; but it is much doubted whether this has not been produced by the decomposition of the chloride of sodium taken in with the food.

In the perspiration, although a volatile acid is perceptible to the senses, and from its odour supposed to be acetic, nothing definite of its real nature is at present known. The juice of the flesh in health is undoubtedly acid, and contains lactic acid, according to Liebig; but its acidity is necessary for the due performance of those chemical changes which take place in the organism. The secretions in rheumatism are no doubt in a defective state; but we have no evidence whatever that altering them will afford any relief to the unbalanced state of the blood, inasmuch as they are only a result, and the cause cannot be reached through a secondary occurrence.

This is a topic which leaves room for much future thought and experiment.

Referring to the case of acids, we may remark that it has been long recognised that the salts of organic acids pass off with the urine as alkaline carbonates; and this they do by giving up oxygen to oxidise the uric acid, which uric acid is certainly not in the blood, for it has never yet been there detected. The object sought, and perhaps obtained, by the use of large quantities of vegetable acids, falls, in my opinion, far short of any real curative measure, since to oxidise the uric acid, which is only one consequence of the disease, can have but little effect on the disease itself.* This argument is still more cogent when we find that the secreted acid passes readily off from the organism. We must, therefore, repeat that acids and alkalis are included in the same list of remedies, applicable more to effects than to causes, useful, it may be, as palliatives, but leaving the original disease unapproached. The favour they have partially obtained is too much in accordance with a practice which we are obliged at this time especially to acknowledge and avoid. This is no less than an attempt to meet only particular trains of symptoms, to the disregard of all the primary and comprehensive characteristics of the disease, and then to

* No equation has been given for disposing of the remaining elements of citric acid, if we give up the oxygen to oxidise the uric acid.

offer to the notice of the profession those means so narrowly directed in the guise of general curative measures. To look no further than the correction of a faulty secretion is a part of that mistake which induces blindness to the real fundamental pathology of disease, while the sight is wholly occupied with the affection of some particular organ. The evil has usurped a wider and still more pernicious influence by especial organs having been brought forward into notice, and the derangement of their functions taken up as central points to which diseases of all kinds are sought to be referred. There is no limit to the opportunity of indulging in this mistake, because there are few diseases in which the primary cause of the disorder, through which the system has been thrown out of the healthy state, does not reach to every organ and its function.

The unfortunate extent to which this propensity characterises our investigations can only be corrected by that enlarged view of causes and consequent simplification of classes of disease, which is at last becoming a more acknowledged object, both of desire and research. I am sure all present feel with me that to pursue that object energetically is the only course to elevate the science of medicine, so that it may keep pace with the expanding attainments of the other sciences.

The action to be ascribed to nitrate of potassa, consists in the mechanical effect of endosmose along

the canal, and its own free elimination by the kidneys, of which it certainly increases the secretion. It produces a sort of minor depletion by drawing off secretion from the blood; therefore in all inflammatory diseases its great value is well known and acknowledged. It may also be remedial as an oxidising agent, because, as it readily parts with its oxygen, it may assist in converting the uric acid in excess into urea and carbonic acid. One reason assigned for its use in rheumatism is, that it assists to replace the salts which are known to be missing in inflammation; but the salts observed to be defective in the blood and urine consist chiefly of chloride of sodium, which is absent in consequence of the abstinence from food, the medium of its habitual supply.

It remains to say a few words on the use of counter-irritants, and of their effects on that more intense form of inflammation which is localized within the chest. The object proposed by blisters is to check the exudation of lymph, which is the result of that intense form of inflammation; and there is great doubt how far the pain and irritation which are set up by the remedy may do more harm than good. We have already noticed that Andral and Gavarret have recorded a case of inflammatory fever (in which of course there was a fibrinated state of the blood) set up in consequence of a severe burn. We should hence conclude that the pain and irrita-

tion which are produced by the action of cantharides are calculated to exacerbate the state of the blood.

A degree of temporary relief may be immediately obtained by drawing off some portion of lymph at the surface, yet the tendency to increase the already disturbed balance renders it a very dubious mode of action. When the system is once quieted, that is, the fever subdued and the balance restored, and the only desideratum is to assist in conveying off the effusion, blisters may be valuable.

In young persons, as we have already noticed, the blood contains a larger proportion of fibrine, which accounts, in some degree, for their circulation being more easily excited. Hence we may deduce, not only a cause for their especial liability in rheumatism to chest complications, but a reason why the application of blisters should be more cautiously used.

I believe that I have now adverted to nearly all the remedial measures which have been used or proposed for the treatment of rheumatism. We have not examined closely the amount of success which has been claimed by various practitioners for so many opposing plans, though we have calculated the degree of efficacy which might, according to my own views of the disease, be expected from them.

I will now present a summary of the treatment, which, keeping still in view the nature of the causes of derangement, would appear most calculated to afford relief. It will then remain for each present

to add for himself such testimony as his own experience may afford to the truth of the result. Of course my own observation in practice bears satisfactory evidence to my own mind, that such plans as I should expect to find most successful have actually been so.

Considering the ascertained continually-ascending scale of the excess of fibrine, up to a certain point, in spite of any amount of loss of blood, we should be led to reject full and successive bleedings as exhausting the strength of the patient in larger proportion than the degree of relief which might be expected by the removal of the fibrine. When we do have recourse to bleedings, sparingly, or at least very cautiously, we should propose a distinct object, viz., to diminish the pressure in the vessels of the joints; also to lessen the rapidity of the circulation, checking in that rapidity the continual over-production of fibrine.

As the surface blood contains a larger relative proportion of fibrine, and as we have assumed that a certain amount of delay is actually present in the vessels of the joints, we should expect that local bleeding would prove a valuable adjunct to that restorative process which we suppose to be going forward in the existing stasis of the vessels.

Having established the indirect method by which the fibrine of the blood may be drawn off by purgations, without that reduction and dangerous state

of depression which would be caused by the simultaneous loss of the blood corpuscles in bleeding, we shall acknowledge the great value of this line of treatment, and only regret the more that the circumstances of the patient present insurmountable difficulties at the time when the attack is most acute. And I think that we should not be disposed to follow the example of some who calculate that the infliction of immediate suffering is compensated by the relief which follows. Natural feeling dictates the rejection of all plans that are in themselves distressing, except in cases when the end cannot be otherwise attained. The value of mercury in rheumatism is only great when the vital organs are the seat of the attack; its chief use, then, is to modify the blood and check the exudation of lymph. In inflammation of the joints, which is a lower grade, and as a general rule unaccompanied by the exudation of lymph, so powerful an engine as mercury is not required, at least, not to be used to its constitutional effect.

I quite, however, acknowledge the prudence of a moderate use of mercury, so as to be ready to extend the effect rapidly, should the emergency of the chest complication call for the full working of the remedy; an extension which occurs so often that it is wise to be forearmed to meet it.

I especially advocate its use during the complications, with a view, not only to immediate recovery, but to the probable diminution of structural damage,

so reducing the chances of permanent foundation for future mischief.

It may, perhaps, cause regret or disappointment, that on the subject of treatment I have no specific plan to advocate; but those who (whether they do or do not agree with me) have heard that my views of the disease reject all special agency, and refer its nature to a particular modification of wide and generally understood causes, will not wonder that I should not propose to meet it by other than the acknowledged plans of treatment which are already known to influence those causes. All the practical good which I hope to derive from what I consider truer views of the disease is to use those acknowledged means according to their proper aims. On the subject of specifics it has long been my conviction, that it is not new remedial agents that we have to seek for, but a juster discrimination of the cases in which to apply those we already possess. The empiric seeks the weapons, we seek the judgment how and when to use them. I never feel more humbled than when I hear any regular practitioner proposing to try the efficacy of this or that plan. It ought to be for us not to try plans, but to know disease, and then use plans with confidence. Those who have but to please the multitude may try experiments; with us, practice must follow, not precede philosophy.

Great as has been the progress towards that true

philosophy, and much as has been done by our own contemporaries and fellow-countrymen, I still believe that an æra in the science of medicine has yet to begin, in which not only will the processes of nature be still more closely traced, but in which the laws which govern aberrations from the normal state will be found to be more simple and comprehensive than has yet been supposed. Several vigorous efforts have indeed been made in this direction, but they have not been sustained or followed up with that steadiness which insures success.

If we inquire the cause of this failure, certainly not due to any want of intellectual energy in the medical profession at large, we shall find it, I fear, in some defect of moral courage.

Theorizing has been degraded to unworthy purposes, and in advancing a novel hypothesis, however well grounded, men of solid attainments and established character fear to be confounded with shallow pretenders. It is no wonder that when used by such men as the disciples of Hahneman, for example, the very word system should have passed into a byword; standing as it does with them to represent a so-called science, which substitutes imaginary positions for physical phenomena, and a sort of metaphysical wandering for solid reasoning. Again, I fear that we may all, at times, have felt that the desire for truth is second to that for the credit which its discovery may earn; and we all know that the penalty of discredit for a

false hypothesis is more unfailing than the meed of honour for a true one. These doubts and fears—moral refrigerators of scientific ardour—may be recognized as hindrances to profound and speculative research; yet we see other countervailing causes at work, from which we may hope well for its future advance. Mechanical means for examination and analysis are improved and multiplied: results more duly classified and recorded; and materials so obtained become public property, and are at the service of future travellers in the same path of inquiry. Increased facilities for intercommunication make men of science, of different countries, better known to each other, and the literature of various nations now equally belongs to all. Thus all around us seems at once to minister abundant materials for scientific induction, and to supply powerful and varied stimulus to the intellect. That these advantages will not be lost on the members of our profession is not my hope only, but my earnest trust.

Whoever may lag in the onward march of knowledge, whether theoretic or practical, they will press to the van, and their courage to pursue truth will daily strengthen with the increasing prospect of obtaining it.

LONDON:
PRINTED BY W. CLOWES AND SONS, STAMFORD STREET.